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CONTENTS

THE WHOLE OF THE LITERARY MATTER IN THE LANCET IS COPYRIGHT

ADDRESSES AND ORIGINAL ARTICLES

Nutrition and Nutritional Diseases. By Prof. LEONARD G. PARSONS, F.R.C.P. II—Rôle of Vitamin C in Disease (*with charts*) 123

Effect of Œstrogenic and Other Sex Hormones on the Response of the Rat to Vasopressin. By F. B. BYROM, M.R.C.P. (*illus.*) 129

Cavernous Hæmangioma of the Orbit Successfully Removed by Krönlein's Operation. By H. B. STALLARD, F.R.C.S. (*illus.*) 131

The Normal Level of Plasma Bilirubin. By JANET M. VAUGHAN, M.R.C.P., and G. A. D. HASLEWOOD, Ph.D. (*with chart*) 133

The Heart in Diphtheria. By H. M. LEETE, M.D. (*with charts*) 136

Outbreak of Acute Respiratory Infection in a Neonatal Ward. By ALICE E. DICKIE, M.B. 139

CLINICAL AND LABORATORY NOTES

Application of Pressure in the Injection Treatment of Hæmorrhoids and Prolapsus Ani. By E. F. ST. J. LYBURN, M.B. (*illus.*) 141

Pneumo-œsophageal Fistula. By BERYL E. BARSBY, M.B., and DOROTHY M. VAUX, M.D. (*illus.*) 142

MEDICAL SOCIETIES

Society for the Study of Inebriety: Drug Addiction in Britain 143

INDEX NOTICE

THE LANCET, Vol. II, 1937 164

REVIEWS AND NOTICES OF BOOKS

British Encyclopædia of Medical Practice. Vol. VI. Under the general editorship of Sir Humphry Rolleston, M.D. Assisted by Prof. F. R. Fraser, F.R.C.P., and others ... 145

Malaria. By Profs. B. Nocht, M.D., and M. Mayer, M.D. 145

Fundamentals of Anatomy. By Carl C. Francis, M.D. 145

The Subnormal Mind. By Prof. Cyril Burt, D.Sc. . 145

Synopsis of Genito-Urinary Diseases. By Prof. A. I. Dodson, M.D. 146

The 1937 Year Book of Urology. By J. H. Cunningham, M.D. 146

Biology for Medical Students. By C. C. Hentschel, M.Sc., and W. R. I. Cook, Ph.D. .. 146

Medicine. By G. E. Beaumont, F.R.C.P. 146

LEADING ARTICLES

PREGNANCY TOXÆMIA AND THE PITUITARY 147

HÆMATOMA OF THE RECTUS ABDOMINIS 147

B.B.S. AND DISEASE 148

TUBERCULOUS MENINGITIS 149

ANNOTATIONS

Raw Apple Diet and Infantile Diarrhœa 150

A Secret Operation 150

The Immunising Antigens of Shiga's Bacillus 150

The Increase in Longevity 151

Concerning Mumps 151

Care of Mental Disorder in London 152

Value of Total Opium Alkaloids 152

Investigation of Asthma .. 153

Are Prosecutions for Poisoning Fair? 153

The New St. George's 154

GRAINS AND SCRUPLES

From a Bacteriological Back-number (III)..... 159

SPECIAL ARTICLES

Clinical Examination and Diagnosis in Cardiovascular Disease. By TERENCE EAST, F.R.C.P. 155

United States of America: Neonatal Mortality 156

Medicine and the Law: New Public Health Act Offence—Duty to Notify Puerperal Fever 157

An International Review of Infectious Diseases: Yellow Fever — Typhus Fever—Small-pox and Vaccination 158

Protection of Eyesight ... 160

Croydon Typhoid Inquiry: Evidence of the Consulting Analyst—Evidence of the Town Clerk—Evidence on Behalf of the Typhoid Outbreak Committee — Evidence of the Medical Officer from the Ministry of Health —Counsel's Concluding Statements 165

THE LANCET 100 Years Ago 144

CORRESPONDENCE

The London M.B. Final (Sir E. Graham-Little, F.R.C.P., M.P.) 161

Early Treatment of Cataract (Dr. Christie Reid) 162

Child Discipline (Dr. R. Miller) 162

Royal Medical Benevolent Fund (Sir Thos. Barlow, F.R.C.P., F.R.S.) 163

Hire-purchase Research (Dr. R. Forbes) 163

Melanosis Coli (Sir A. F. Hurst, F.R.C.P.) 164

Who Pays the Doctor Called to an Accident? (Mr. Harcourt Kitchin).. 164

PUBLIC HEALTH

Prevalence of Dysentery .. 172

Infectious Disease in England and Wales 171

OBITUARY

Thomas Tendron Jeans, M.B. 172

William L. M. Gabriel, M.B. 172

MEDICAL NEWS

University of London 173

Royal Institute of Public Health and Hygiene ... 173

B.M.A. Scholarships and Grants in Aid of Research 173

Society for Constructive Birth Control 173

Institute of British Surgical Technicians 173

Swedish Woman Professor of Medicine 173

The Film Biology 173

State Veterinary Service .. 173

British Postgraduate Medical School 173

Society for Relief of Widows and Orphans of Medical Men 173

Course on Mental Deficiency 154

Royal Medical Benevolent Fund 164

German Journal for Rheumatism 172

National Institute of Economics and Social Research 172

NOTES, COMMENTS, AND ABSTRACTS

Doctor and Patient. By Sir MAURICE CASSIDY, F.R.C.P. 175

Serotherapy in Leprosy .. 179

A Study of Racial Differences 179

Protection against Measles 179

New Preparations: "Nestrovite" Brand Vitamin Concentrate—"Wellcome" Brand Calcium Mandelate Compound — Sulphanilamide Tablets G.L. 179

Medical Diary 174

Births, Marriages, Deaths 174

Vacancies—Appointments. 180

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II—RÔLE OF VITAMIN C IN DISEASE

All the vitamins have some function to perform in the chemistry of the cell, and in their absence, although the cells concerned may still survive and be capable of growth and multiplication, they exhibit structural changes. The particular cells involved vary with the different vitamins; thus, the specific effect of vitamin A is on epithelial cells, that of the B complex on the nerve-cell; vitamin C affects all supporting tissue cells and D the cartilage cells. Vitamin C has, in addition to its specific action of regulating the colloidal condition of intercellular substance in all mesenchymal structures, another important specific function in the body—a respiratory one—since it forms an oxidation-reduction system in the cell. Hence a deficiency of it may be the basis of many pathological changes, and it is not surprising that this has been invoked as the cause of many disease processes, especially those connected with the hæmorrhagic states. Many of these suggestions are so inaccurate as to be utterly absurd. There is a deplorable amount of what the Americans call "commercial ballyhoo" concerning the vitamins, but the claims put forward for them must be investigated because sometimes things revealed to babes are hidden from the wise. (The 1929 epidemic of psittacosis owed the discovery of its origin to a chance remark of a young child that "I know what is the matter with auntie—it is what the parrot has," knowledge which until then had been denied to a colleague of mine.) I propose to look at the less fantastic of these claims to see if there is any evidence that vitamin C is concerned in any other condition than frank scurvy.

On an adequate diet, breast milk contains 0·06–0·08 mg. of vitamin C per c.cm. which is more than enough for the needs of the infant (20 mg. ascorbic acid daily), but on inadequate diets it may sink as low as 0·01–0·02 mg. per c.cm. (Selleg and King 1936). Fresh cow's milk contains a third to a sixth the amount present in breast milk, and further amounts are lost by pasteurisation, ageing—especially if exposed to light—and by reheating. Cow's milk also contains an oxidase that is able to convert the vitamin to its oxidised and inactive form, and pasteurisation instead of accelerating oxidation may sometimes inhibit it by destroying more of the oxidase than the acid. The presence of copper in small amounts, as by storage in copper vessels, also increases oxidation. Sharp (1936) gives the following values for ascorbic acid per litre of cow's milk:—

Fresh milk	20·1 mg.
After three days at a temperature of 2° C. ..	11·3 „
After pasteurisation at 62°–63° C. for 30 minutes	11·0 „
After pasteurisation if copper is present	1·7 „

Of the vitamin-C intake, a considerable proportion is lost in the stool, some in the urine, some destroyed in the alimentary tract, and the rest stored and utilised. There is no evidence of any serious loss during absorption. The vitamin is stored in the highest concentration in the pars intermedia of the pituitary gland; high concentrations are also found in the anterior lobe of the pituitary, the adrenals—particularly the cortices—the corpus luteum, the interstitial cells of the testes, the tonsils, and to a lesser degree in the liver, spleen, &c. Many of the organs in which it is present in largest amount are associated with sex changes, and it has been shown (Glick and Biskind 1936) that the fluctuation of vitamin-C content of the corpus luteum parallels the changes in the amount of luteal hormone—progesterone—in the gland, an association which may prove to be of considerable significance. The vitamin is present in the blood and urine in its reduced form and in the tissues in its reversibly oxidised inactive form—dehydro-ascorbic acid.

Certain animals, for instance the cow, rat, mouse, and fowl, can synthesise vitamin C and therefore do not require it in the diet. Röhmer and Bezssonoff (1935) claim that the human infant under a year old should be added to this list of animals; this I find it impossible to accept. It is the capacity for synthesising vitamin C that accounts for its low percentage in cow's milk.

Gross deficiencies of vitamin C lead to the development of scurvy, but occasionally, as I pointed out in the first lecture, scurvy occurs even when the diet contains considerable quantities of vitamin C; an infant admitted to the Children's Hospital with frank scurvy was having daily orange juice containing 60 mg. of ascorbic acid. We have suspected that this might be due to lack of absorption, though we have not been able to demonstrate this chemically. Indeed, in the case to which I have just referred, cure was actually obtained by giving large doses of ascorbic acid by mouth. It is probable that some children require more vitamin than others.

Until vitamin C was synthesised the human race was dependent upon the annual crops of fruit and vegetables for its antiscorbutic food, a veritable hand-to-mouth existence that led to severe epidemics of scurvy, as in Ireland and Norway when the potato crops failed. It has been stated that this synthesis "may have more far-reaching effects on the welfare of the race than the work which has been done on all other vitamins put together."

The time required for the development of frank scurvy on an inadequate diet in the infant is about 6 months (2–9 months Park, Guild, Jackson, and Bond 1935), but usually only from 4–8 weeks in the adult. This difference is said to be due to the fact that the diet of the adult sufferer is more likely to be free from vitamin C than even that of a child on pasteurised milk. There are, however, at least two other possibilities: first, that as with thyroxin and vitamin D, the new-born baby starts with a store of vitamin C obtained from its mother during pregnancy; secondly, the effect of trauma in determining the occurrence of certain symptoms of scurvy is much more likely to be a factor in the adult than the infant. If trauma is ever a factor in the onset of infantile scurvy, it is probably self-inflicted. Despite the long latent period the symptoms of acute infantile scurvy appear suddenly and it is much more probable that some infection, perhaps only slight, is the real exciting factor—the onset of an infection fanning the smouldering embers of the latent disease into the blazing fire of frank scurvy. A similar state of affairs occurs in another vitamin-deficiency disease—tetany. A child with symptomless latent tetany contracts a slight infection and tetany becomes manifest

* Withering lectures (abridged) delivered before the University of Birmingham on May 11th and 13th, 1937.

as a convulsion. An interesting parallel to the sudden onset of the symptoms in acute scurvy is presented by their rapid cure when large doses of ascorbic acid are administered; the paralysed child crying with apprehension and pain will in 48 hours move its legs freely and allow them to be handled almost with impunity.

Latent Scurvy

Admitting the existence of a long latent period in scurvy, the question has often been asked whether it is possible by clinical signs and symptoms or by chemical tests to determine the presence of a condition which has received various designations—latent scurvy, sub-scurvy, scorbutic dystrophy—exactly as latent tetany can be diagnosed by clinical and chemical tests. The conception of latent scurvy was first emphasised by Alfred Hess (1917) of New York, who pointed out that although this condition could be diagnosed only by the response to specific therapy, yet certain symptoms might be suggestive—e.g., if children who had been fed for a considerable period on pasteurised milk, at the age of about 6–9 months suffered from loss of appetite, ceased to thrive, lost their healthy appearance, became pale, had increased knee-jerks, and possibly a rapid pulse and respiration.

ASCORBIC ACID IN URINE

A few years ago Tillmanns of Strasbourg devised a test to distinguish between lemon juice made from lemons and that produced synthetically; the fresh natural juice discharged the blue colour of 2:6-dichlorophenolindophenol whereas the artificial product did not. Later it was found that this effect was due to the presence of ascorbic acid and this test, which can be used quantitatively as well as qualitatively, has been extensively applied to the urine by Harris and his colleagues (1935) as a method of detecting latent scurvy. Although other substances present in the urine—e.g., glutathione, ergothionine, cysteine, thiosulphate—will decolorise the dye, yet in ordinary circumstances the greater part of the reducing substance present in the urine is ascorbic acid. To eliminate these interfering substances, a simple modification of the test was suggested by Prof. Haworth, depending on the fact already mentioned that small amounts of copper catalyse ascorbic acid and render it inactive. If, therefore, two separate estimations of the amount of reducing substances present in the urine are made, the one after boiling with a small amount of copper sulphate and caustic soda, the difference between the two results indicates the amount of ascorbic acid excreted. Another method is that in which the interfering substances are precipitated by mercuric acetate and then H_2S is passed through the urine to convert all the ascorbic acid to the reduced form before carrying out the titration (van Eekelen 1936).

Harris and his colleagues (1935, 1936) have made certain statements in regard to the results obtained by their test:—

1. Normal individuals appear to have a reserve supply of vitamin C, since they excrete in the urine at an almost steady rate when the supply in the diet is cut off.
2. The urine of normal adults shows an almost constant daily output of 30–33 mg. and that of the young child 1–2 mg.
3. When a large dose (100 mg. for infants) is given to a normal person there is a considerable rise in the amount excreted in the urine, because he is saturated with the vitamin and the degree of response to such a dose indicates the degree of saturation.
4. In a scorbutic patient the amount of ascorbic acid excreted is diminished and there is no increase after the test dose, but after cure or saturation of the tissues there is the same peak in the excretion as in a normal individual.

Using this test he claims to have proved the existence of many examples of latent scurvy, a condition in which the amount of ascorbic acid excreted is less than normal, and the peak after the test dose shows "unsaturation," which can be converted into "saturation" by adequate amounts of vitamin C. According to his latest figures, an adult, weighing 10 st., who excretes less than 13 mg. of ascorbic acid daily and fails to give a "good response" on the first or second day to a test dose of 700 mg. may be presumed to be taking in his diet less than the minimal requirements of vitamin C—i.e., 25 mg. of ascorbic acid or 45 c.cm. of fresh orange juice ("the greater part of the juice of a moderate sized orange.")

This test and Harris's figures have been generally accepted as the basis for determining the existence or otherwise of latent scurvy. The uniformity of daily excretion has, however, not been found by everyone. Johnson and Zilva (1934) found that there was no definite normal amount of ascorbic acid excreted in the urine daily, and that it might even reach 70–150 mg.; further, that although some were so unsaturated as to require doses of 100–150 mg. of ascorbic acid daily for a "considerable time" before saturation occurred, yet they were without any symptoms of ill health. Using the term "saturated" as meaning the more or less constant daily output of ascorbic acid in the urine after a constant daily intake of ascorbic acid, Zilva (1936) states that there is no clinical nor laboratory evidence that unsaturated persons are in a worse state of health than saturated ones. He also points out that in estimating the balance between the intake and the output of ascorbic acid the amount unaccounted for varies not only with the size of the test dose and the subject, but also at different times with the same individual on the same diet, and that there appears to be a "wide margin of 'unsaturation' which has no obvious detrimental effect on the individual." To apply to this zone of "unsaturation" such expressions as "hypovitaminosis C," "latent or potential scurvy," "vitamin C sub-nutrition," "sub-acute scurvy," expressions that imply ill-defined pathological conditions, is, in his opinion, not justified by the present state of our knowledge.

We have used the test dose method frequently in infants and children, but have given a much larger amount (500 mg.) than that suggested by Harris (100 mg.) and repeated the dose if necessary until saturation occurred. Our definition of saturation is the excretion of not less than 10 per cent. of the test dose—i.e., not less than 50 mg. Without being aware of Zilva's criticisms we had come to the conclusion that although such a test dose might be helpful in determining the presence of latent scurvy, Harris's standard for the normal excretion of the urine in infants was not always correct, and that the figures obtained for excretion alone were too variable to be of any great value (see Fig. 1). It is true that with very low excretion values, the diagnosis of latent scurvy might be accepted, but many cases that are classed as such excrete amounts of ascorbic acid which, although small, are not less than those excreted by normal children. Also, we have found that in frank scurvy the first effect of a test dose of vitamin C is to *diminish*, not to increase, the amount excreted in the urine, an observation that may be used as evidence of the presence of scurvy. Again, and bearing out Zilva's view, we have recently had a child with severe scurvy whose symptoms were

completely cured by large doses of ascorbic acid, but who, even after considerable periods of apparently perfect health, was "unsaturated."

We cannot however, as Zilva does, dismiss the existence of latent scurvy, if for no other reason than the good effects of administering vitamin C to infants

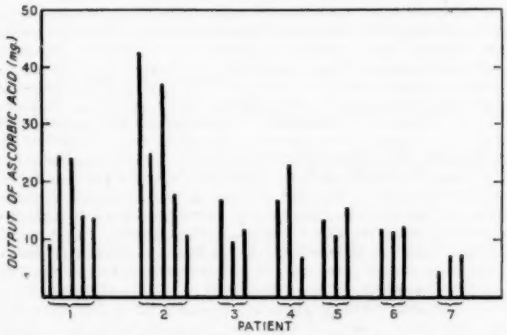

FIG. 1.—Variations in output of ascorbic acid on successive days in seven patients on an ordinary ward diet.

with vague symptoms of ill health. We therefore have come to the conclusion that the test, particularly as modified by us, does bear out the contention of Hess that there is a condition of latent scurvy analogous to latent tetany.

ASCORBIC ACID IN THE BLOOD

Ascorbic acid is present in the blood in its reduced form, but should there be hæmolysis dehydro-ascorbic acid is formed most probably by the oxyhæmoglobin set free, although after the initial hæmolysis further oxidation is prevented by the disorganised corpuscles (Kellie and Zilva 1936). Moreover the blood, like the urine, also contains other reducing substances. If therefore it is desired to obtain accurate estimations of the amount of ascorbic acid in whole blood, treatment with mercuric acetate and H_2S should be carried out (van Eekelen 1936). In serum the vitamin can be estimated (Abt, Farmer, and Epstein 1936) without these procedures, but in view of the possibility of some hæmolysis having occurred, it is also better with serum to carry out the mercuric acetate and H_2S procedure. It is possible to form an idea of the state of saturation of an individual by determining the ascorbic acid either in the urine or the blood, preferably the latter according to van Eekelen since a single examination only is needed and because the daily output in the urine varies greatly in the same person and from person to person even with about the same degree of saturation. The importance of using fasting specimens for the estimations of blood ascorbic acid has been stressed by Greenberg, Rinehart, and Phatak (1936) because they found that the level in the plasma rises from 15 per cent. to 60 per cent., 2–4½ hours after the ingestion of 6 oz. of orange juice. These workers agree, with others, that the blood ascorbic acid in the "normal" individual parallels the vitamin-C intake, and that fasting levels ranging between 0·7 and 0·9 mg. per 100 c.cm. appear to be adequate, whereas those below 0·7 are probably suboptimal and below 0·5 mg. per 100 c.cm. must be considered low.

EFFECTS OF DIET ON OUTPUT

It has been suggested that the amounts of any vitamin required depend to some extent on other factors in the dietary.

Perhaps the best known example of this is Mellanby's insistence that cereals, particularly porridge, contain something which antagonises vitamin D and which is an actively decalcifying agent and that, therefore, the child who takes any considerable quantity of cereals in the diet requires more vitamin D than the child who has only a minimal amount. The child fed on cow's milk requires more vitamin D than the ordinary breast-fed child because of the better ratio of calcium and phosphorus in breast as compared with cow's milk. It is also stated that the greater the carbohydrate content of the food in man, the greater the need for vitamin B_1. At any rate, in animals the amount of B_1 required depends upon the body-weight and the caloric intake. If large quantities of carbohydrates or alcohol are taken, therefore, the need for vitamin B_1 is increased. In a recent experiment ten patients with alcoholic polyneuritis were given one pint to one quart of pure blended whisky (caloric value 1400 to 2800) daily for from 2 to 18 weeks, with a generous diet and generous amounts of vitamin B_1, and in every case the polyneuritis was relieved (Strauss 1936).

Two papers (Ahmad 1936) have recently been published showing that vitamin-C output in the urine is increased by taking a high protein or a high fat diet. In these investigations Harris's test was used, but no attempt was made to eliminate the effect of other reducing substances in the urine. Heinemann (1936) who repeated these experiments did eliminate them and thereby found that the output of vitamin C is quite uninfluenced by the amount of protein (or fat) in the diet, but that the total reducing power of the urine did vary directly with the quantity of protein in food, and that the reducing power of the urine depended chiefly on the output of thiosulphate; he also found that cystine taken with a low protein diet had the same effect as large quantities of protein on the total reducing power of the urine, and in his opinion the sulphur-containing amino-acids are the source of the reducing body (thiosulphate) in the urine.

These results, therefore, demonstrate another fallacy that must be considered in the clinical use of the Harris-Ray test for urinary ascorbic acid.

Cataract

Another interesting clinical parallel with tetany and with scurvy has recently been traced with regard to the occurrence of cataract. It is well known that there is a form of cataract in prolonged states of hypocalcæmia; thus it has been found in association with tetany and in adults who have suffered from cœliac disease since childhood. The aqueous humour contains 20 mg. per 100 c.cm. of (reduced) ascorbic acid, about twenty times that present in the blood, and the lens itself contains even more, 30 mg. per 100 c.cm. (Campbell 1936). During the development of cataract the vitamin gradually disappears from the lens and aqueous humour and eventually is found only in the capsule. Opinions vary as to whether these changes are the cause or the result of the development of cataract. It is stated that the vitamin can diffuse from the blood into the aqueous humour and from the latter get into the lens, but that the reverse process cannot occur, and hence the vitamin content of the lens is higher than that of the aqueous humour. If anything increases the permeability of the barrier—e.g., uveitis, old age—vitamin C, which is necessary for the internal respiration of the lens, disappears from the aqueous and the lens becomes opaque. Gowland Hopkins first pointed out that the lens being separated from the blood stream can only take up the oxygen necessary for its metabolism by an oxidation-reduction system, and showed that such a system was found in glutathione, a compound which also disappears from the lens during the development of a cataract.

According to Hopkins, these two substances—ascorbic acid and glutathione—are the most active known reducing substances in living tissue. It may be mentioned that glutathione completely protects ascorbic acid from oxidation by copper catalysis and by "hexuronic oxidase" and it has been thought that vitamin C can be derived from the reduction of sugar by glutathione (Hopkins and Morgan 1936).

Although the administration of ascorbic acid to patients suffering with cataract has not been found to have any influence on the development of the cataract, it is of interest to note that the disease is most frequent in the poorer classes whose diet is probably deficient in vitamin C.

Gastric Ulcer

Recently, Harris and his colleagues (1936) have estimated the vitamin C in the urine of voluntary hospital patients and found that amongst the lowest excretions "a quite disproportionate number were of patients with gastric and duodenal ulcer." It is not unimportant in this connexion to remember that in guinea-pigs a deficiency of vitamin C may predispose to gastric ulcer (Smith and McConkey 1933).

The most important contribution to this subject is that by Archer and Graham (1936).

At St. Bartholomew's Hospital it was found that in three-quarters of those who died of a general peritonitis after operation on the stomach the infection appeared to be due to "a bacterial leakage at the site of the anastomosis as a result of an almost complete absence of any fibrinous response along the suture line." Remembering that the wounds of patients with scurvy healed badly, it occurred to Archer and Graham that the cause of this lack of healing might be due to depletion of the ascorbic acid in the body. They therefore investigated by the Harris technique 9 patients suffering from duodenal ulcer who were taking a modified Lenhartz diet and in 6 found definite evidence of sub-scurvy.

Although the effect of scurvy in retarding the healing of wounds and ulcers renders it imperative that the diet used for gastric and duodenal ulcers should contain vitamin C in sufficient amounts to saturate the individual, the usual "ulcer diets" are quite inadequate in this respect. Two ounces of orange juice daily is sufficient to prevent the development of sub-scurvy, but Archer and Graham think it wiser to give 1000 mg. of ascorbic acid on three successive days (to ensure saturation) either by mouth or, if the patient is vomiting, intravenously, and to follow this up with two ounces of fresh orange juice daily.

Acute Rheumatism

It is well known that adults and children of the upper, and therefore presumably better fed, classes escape rheumatic infection, and the suggestion has from time to time been made that the resistance to that infection may be in part a matter of nutrition.

In 1935 Warner and Winterton investigated the diets of rheumatic children and those of adequate controls and came to the conclusion that no one factor could be incriminated as a contributory cause for the occurrence of rheumatism. They also noted that the consumption of fresh fruit and vegetables by the rheumatic families and children was much higher than that of children in poor-law schools and at Christ's Hospital, in which institutions there was a low incidence of rheumatism.

Despite these findings, it is not surprising that in this age of vitamin C, the question as to whether there is any association between vitamin C and rheumatism has been the subject of some discussion.

Rhinehart (1935), who is the chief protagonist of the view that rheumatic fever may result from the combined influence of vitamin-C deficiency and infection, infected guinea-pigs with various strains of beta streptococci and other organisms. In the presence of adequate vitamin-C nutrition rheumatic lesions were not observed, but when superimposed on vitamin-C deficiency the infection produced a valvulitis with considerable frequency, changes in the myocardium not infrequently, and subcutaneous nodules occasionally.

Sendroy and Schultz (1936) have found that patients with rheumatic fever may develop a real hypo-vitaminosis on an ordinarily adequate diet, in that 8 of 13 cases when compared with controls showed an apparent ascorbic acid deficiency. They suggest that it is much more reasonable to regard such deficiency as the result of an infectious process rather than as initiating the infection. Schultz (1936) repeated Rhinehart's experiments but considered that the lesions obtained were not similar to those found in acute rheumatism. He also found that giving ascorbic acid to rheumatic children had no effect on the incidence of recurrent attacks, nor did the administration of large doses of ascorbic acid or orange juice produce any demonstrable effect on the course of the disease. The most recent work on this subject is by Taylor (1937) who concludes that the one specific factor associated with the cardiac lesions in guinea-pigs is scurvy and that extraneous infection increases neither the incidence nor the severity of the lesion, also that "the lesions resemble those of rheumatism only in that they are both a diffuse non-purulent carditis without gross valvular vegetation."

However, Abbasy, Hill, and Harris (1936) have recently produced evidence in support of Rhinehart's claim. A group of "107 rheumatic cases" and "86 convalescent rheumatic cases" was contrasted with a control group of 64 individuals who were apyrexial, "mostly cases of congenital deformity or cases of debility well on the way to recovery," and also with another control group of "patients with surgical tuberculosis, 23 of which were called active, 19 who had improved under treatment half-active, and 46 quiescent." All these patients were on the same liberal diet containing an ample supply of vitamin C. They found that patients suffering from juvenile rheumatism, both active and convalescent, had a deficit of vitamin C in the urine, which they regard as indicating that there is an increased destruction of the vitamin in rheumatism, so that the rheumatic subject appears to have an increased need for vitamin C. These statements may or may not be true, but on the evidence produced in this paper, exactly the same deductions could be drawn for tuberculosis as for acute rheumatism. Indeed, if the results given for "active rheumatics" be compared with "active surgical tuberculosis," "convalescent rheumatics" with "half-active tuberculosis," "controls" with "quiescent tuberculosis" the curve of figures for urinary excretion is almost identical in each corresponding group. What criteria were used for determining "quiescence" I cannot say, nor can I visualise what is meant by "half-activity." It may be that a pyrexia occurs in the quiescent group and not in the others. If so, pyrexia—which, of course, may be very slight in acute and absent in convalescent rheumatism—as an expression of an infection appears to be the only common factor in the two diseases to which the diminished output of vitamin C might be attributed. In any case, I submit that the evidence Harris and his colleagues have produced directly negatives the suggestion that they imply—if they do not actually state—that vitamin C has a specific relation to rheumatism. Again the weakness of their case is shown by the fact that in contrast to the careful investigation by Schultz at the Rockefeller Institute Hospital described above, which showed the failure of ascorbic acid as a therapeutic agent in acute rheumatism, the only clinical evidence they produce in its favour is a quotation from the report of a school medical officer to an urban district council, which reads as follows: "Rheumatism is largely avoided if plenty of the citrous fruits are taken, especially if lime or lemon juice is taken in water, along with green vegetables and an increased amount of water. I find that heart trouble of rheumatic origin often disappeared by including the aforesaid in a dietary"—statements which no one with even only a nodding acquaintance with rheumatic carditis could possibly accept.

If, indeed, the rheumatic child has an increased need for vitamin C and destroys more of the vitamin

than the normal child, then surely every case of rheumatism, active or quiescent, should show decreased excretion of ascorbic acid. Actually, we have found that the majority of a group of such children do exactly the opposite and excrete more than controls.

We examined 103 children divided into five groups in two institutions, the Children's Hospital and Moseley Hall. In the hospital, the children were arranged in three groups :—

(1) 18 who were suffering from *acute rheumatism* at the time of their admission.

(2) 12 suffering from an *acute infection* other than rheumatism.

(3) 24 *controls*, most of whom had a surgical complaint, and none of whom showed any signs of infection.

The children in these three groups were given the ordinary generous hospital diet, and those in the first and third groups did not have extra orange juice. At Moseley Hall, two groups of children both receiving the same diet were examined :—

(4) 31 *convalescent rheumatic* children ; such children are transferred from the hospital to Moseley Hall when the acute phase of the disease is over, but, of course, some are subject to recurrent attacks.

(5) A control group of 18 *non-rheumatic children*, convalescent from such conditions as empyema, asthma, bronchiectasis, debility, tuberculosis, nephritis.

The urinary excretion of ascorbic acid of the children varied considerably (Fig. 1), particularly in those who had acute rheumatism, but from the graphs (Fig. 2) it is clear that the majority of the children with acute rheumatism excreted more than the "infection" control group, and that the majority of rheumatic convalescent children excreted more than the convalescent controls. With regard to saturation, 61 per cent. of the hospital non-infectious control children were saturated after the first test dose of 500 mg. of ascorbic acid and an exactly similar proportion of convalescent rheumatic children, whereas of children with acute rheumatism 50 per cent. were saturated after the first dose, practically identical with the proportion (46 per cent.) of control convalescent children.

In an attempt to assess the influence of pyrexia in rheumatism on the urinary excretion, 6 children with chorea were given an artificial pyrexia by the injection of T.A.B. ; the results are variable but sometimes there was actually a slight increase (Fig. 3).

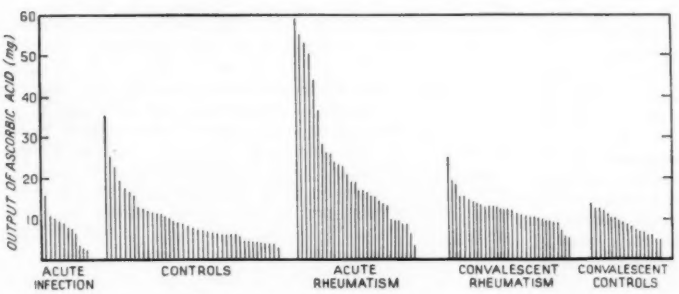

FIG. 2.—Urinary excretion of ascorbic acid showing increased output in children with acute rheumatism and during convalescence.

It is reasonable to assume that vitamin-C stores may be depleted by infection and such might explain Harris's finding, but it certainly will not explain an increased output of ascorbic acid. The question whether *salicylate of soda* will affect this output at once suggests itself, especially since it has been found to precipitate scurvy and death in guinea-pigs receiving diets deficient in vitamin C. Harris and his colleagues do not say whether their patients were being given salicylates, but we presume that they were ; it was certainly being given to some of our patients with acute rheumatism at the time of admission to hospital and to all the convalescent rheumatic children. Another possible factor in these results is *the*

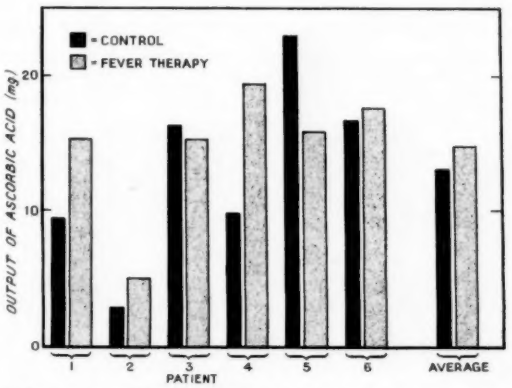

FIG. 3.—Influence of artificial pyrexia (T.A.B.) on urinary output of ascorbic acid in six children.

pH of the urine since it has been shown that the administration of bicarbonate of soda and ammonium chloride in amounts sufficient to alter the *p*H of the urine to 6·5–7·0 and 5·0 respectively produces a significant decrease in the excretion of ascorbic acid in the one case and an increase in the other. We always prescribe either the same or double the amount of bicarbonate as of salicylate of soda with the result that the urine usually has a *p*H of 7·0 whereas the rheumatism itself probably increases the total amount of acid in the urine. Recently, Daniels and Everson (1936) found that the administration of small amounts of aspirin almost doubled the excretion of ascorbic acid. We have repeated these experiments giving 50–75 grains of aspirin with double the amount of bicarbonate of soda daily, and in most instances the ascorbic acid excreted was found to be slightly increased.

The excretion of ascorbic acid in the urine, therefore, probably depends upon the sum total of the following, and possibly other, factors : the severity of the rheumatism ; the amount of salicylate and bicarbonate taken ; and—in view of Coombs's (1937) observations on the modification of the *p*H of the urine by simple diets—the effect of the diet. In our cases most of these factors, except the severity of rheumatism, were constant and our results may be explained by the severity of the infection and in some cases by the administration of salicylate. This explanation however cannot account for the results of Harris and his colleagues. The only conclusion therefore that we can draw is that vitamin C does not stand in any ætiological relationship to rheumatism. In spite of this conclusion, one interesting parallel between acute rheumatism and scurvy should be mentioned. The earliest pathological change in rheumatism is the swelling of collagen around small blood-vessels which

afterwards become invaded by fibroblasts with the formation of an Aschoff's nodule, and Wolbach (1936) regards ascorbic acid as playing a late rôle in the formation of collagen. It would seem, therefore, that both scurvy and acute rheumatism are concerned with the metabolism of collagen, but whether there is any chemical difference in the two collagens I do not know.

Though I have been rather critical of the work of Harris and his colleagues, I believe that their test may be a good one and helpful provided precautions are taken, both with the test and with the interpretation of its results. It has certain fallacies—some of which we have been able to deal with; certain of the statements are incorrect, at any rate as far as children are concerned; and the deductions—some of which are probably right—have been far too sweeping and unjustified by the data.

Addison's Disease

Both the medulla and cortex of the adrenal gland have a high content of ascorbic acid and the cortex also contains "hexuronic-acid oxidase"; indeed, it was from the adrenal cortex that Szent-Györgyi originally isolated ascorbic acid. These facts, together with the possible relationship between the parathyroids and vitamin D, have given rise to speculation regarding the relation of vitamin C to Addison's disease. King (1936) maintains that the function of ascorbic acid in the adrenal is general, rather than specific, but two other suggestions as to the cause of the high content of ascorbic acid have been made: first, that it is to keep the oxidation-reduction potential in the adrenals in a correct state and by so doing to stabilise both the medullary hormone (adrenaline) and the cortical hormone (cortin); secondly, that it is to play a part in the regulation of pigmentation of the skin.

Wilkinson and Atkinson (1936) found a state of vitamin-C subnutrition in three cases of Addison's disease, the degree of subnutrition varying directly with the severity of the illness, but they do not think that it is possible to relate this vitamin-C deficiency with lack of its storage in diseased adrenals. They did not find any change in pigmentation during a test period of intensive oral therapy, although others have claimed that the pigmentation of Addison's disease has been relieved by oral and also by intramuscular ascorbic acid therapy. There is certainly an earthy tint in infantile scurvy that disappears when the child recovers, but in my opinion there is a great difference in the pigmentation of the two diseases; that of scurvy is general, whereas that of Addison's disease is greater in some sites—e.g., the flexures—than others; also the pigmentation of scurvy is very similar to that seen in some cases of acute hæmolytic anæmia and of acute osteomyelitis, which I have interpreted as indicating an affection of the bone-marrow.

So far the evidence that vitamin C can control the course or the symptoms of Addison's disease is quite unconvincing.

Dental Disease

In view of the striking oral symptoms in scurvy, the question whether deficiency of vitamin C has any effect on the structure of the teeth has attracted considerable notice. Hanke (1933) claimed that by giving one pint of orange juice and the juice of one lemon daily he was able to cure gingivitis and to arrest 50 per cent. of the caries in a group of children. In frank scurvy the supporting structures of the teeth are affected, leading to gingivitis and progressive loosening changes which, in many respects, are comparable to pyorrhœa. Experimental work on guinea-pigs can only be quoted in a general sense because their teeth differ from those of human beings in that they grow continuously. Nevertheless, experimental and pathological findings and clinical observations do suggest that during childhood, the period when growth of the body and the teeth is most active and "the susceptibility to dental disease is greatest, it is of paramount importance that the diet should include an adequate intake of this vitamin" (Pitts 1935).

Diphtheria

Recently Bamberger and Zell (1936) of Hamburg have reported that better results in children suffering from malignant diphtheria have been obtained by giving vitamin C and cortin in addition to antitoxin than with antitoxin alone. This treatment is based on the following facts: (1) in malignant diphtheria the adrenals show marked pathological changes; (2) in-vitro vitamin C inactivates diphtheria toxin; (3) in guinea-pigs the action of diphtheria toxin can be "overcome" by the administration of adrenal cortex and ascorbic acid together, whereas either alone is ineffective; (4) guinea-pigs on a diet deficient in vitamin C are much less resistant to diphtheria toxin than normal animals; and (5) the administration of vitamin C to normal guinea-pigs increases their resistance to the toxin.

Pneumonia

Pneumonia has, of course, been hailed as an illness which can be improved by administration of large amounts of ascorbic acid, a statement made because a diminished urinary output of vitamin C has been found during the period of the fever, but no relationship has been discovered between the clinical condition of the patient and the vitamin-C content of the urine.

Hæmorrhagic States

In my opinion there is no justification for associating lack of vitamin C with any hæmorrhagic conditions other than scurvy and the anæmia of scurvy. Hæmorrhage in scurvy is not due to any change in the blood, but to failure of the production of the cement substance in the capillaries, and is, therefore, in the nature of an accidental happening. Conditions such as hæmophilia, purpura rheumatica, Schönlein's purpura, thrombocytopenic purpura, myeloid leukæmia, all of which have been claimed to be benefited by the administration of vitamin C, are essentially different from scurvy in their pathology. The hæmorrhages in these diseases are for the most part due to changes in the blood, rarely to some change in the endothelium, but never to the absence of cement substance in the capillaries.

We have investigated a few cases of purpura rheumatica and in none has there been any evidence of vitamin-C deficiency; moreover, Dam (1935) and also Almquist and Stokstad (1935) have produced a disease in chickens characterised by an increased blood clotting time and scurvy-like hæmorrhages which they were unable to cure by giving vitamin C, but could cure by the administration of a fat-soluble substance occurring in pig's liver, hempseed, and certain vegetables which they call "anti-hæmorrhagic vitamin K." Further, Rusznyák (1936) in conjunction with Szent-Györgyi himself, found closely associated in the cell with vitamin C a substance labelled "vitamin P" which he claims has a definite effect in decreasing the fragility of the capillary walls in certain conditions in which ascorbic acid is inefficacious.

Certain of the hæmorrhagic disorders may therefore be due to other vitamins akin to vitamin C.

Conclusions

From this review certain definite conclusions may be drawn:

First, the pregnant woman and nursing mother must have a generous intake of vitamin C in order that the new-born infant shall have an adequate store of vitamin C at birth and receive adequate supplies during infancy, and thereby prevent the occurrence of latent or frank scurvy.

Secondly, the artificially fed child must be given generous supplies of vitamin C.

Thirdly, an infection or pyrexia or indeed anything that considerably increases basal metabolism may either deplete the available body stores of vitamin C, or render it less potent or interfere with its absorption from the alimentary tract, possibly by the occurrence of achlorhydria or alteration of the normal *p*H of the stomach and intestines. Under such conditions more vitamin C is required, just as in pyrexia an increased intake of fluid is necessary for the increased metabolism, and in infections greater quantities of parathyroid hormone, insulin, hæmatinic factor, and iron are required. Nevertheless, there seems to be nothing to support the view that the vitamin stands in a causal relationship to any disease except the various manifestations of scurvy. There is no doubt that, as in other varieties of avitaminosis, where the intake is insufficient infection may be more likely to occur. There is, however, no justification for the administration of large amounts of this vitamin as a therapeutic or even as a prophylactic measure in every variety of infection; indeed, Wright (1936) records the development of tonsillitis, otitis media, and abscesses in patients who were completely "saturated" by daily intravenous doses of 1000 mg. of ascorbic acid for many days.

REFERENCES

Abbasy, M. A., Hill, N. G., and Harris, L. J. (1936) *Lancet*, **2**, 1413.
— Harris, L. J., Ray, S. N., and Marrack, J. R. (1935) *Ibid*, **2**, 1399.
Abt, A., Farmer, C. J., and Epstein, J. M. (1936) *J. Pediat.* **8**, 1.
Ahmad, B. (1936) *Biochem. J.* **30**, 11.
Alquist, H. J., and Stokstad, E. L. R. (1935) *J. biol. Chem.* **111**, 105.
Archer, H. E., and Graham, G. (1936) *Lancet*, **1**, 710.
Bamberger, P., and Zell, W. (1936) *Z. Kinderheilk.* **58**, 307.
Campbell, D. (1936) *Brit. med. J.* **2**, 1133.
Coombs, H. I. (1937) *Lancet*, **1**, 1043.
Dam, H. (1935) *Biochem. J.* **29**, 1273.
Daniels, A. L., and Everson, G. J. (1936) *Proc. Soc. exp. Biol., N.Y.* **25**, 20.
van Eekelen, M. (1936) *Biochem. J.* **30**, 2291.
Glick, D., and Biskind, G. R. (1936) *J. biol. Chem.* **113**, 27.
Greenberg, L. D., Rhinehart, J. F., and Phatak, N. M. (1936) *Proc. Soc. exp. Biol., N.Y.* **35**, 135.
Hanke, M. T. (1933) Diet and Dental Health, Chicago.
Harris, L. J., and Ray, S. N. (1935) *Lancet*, **1**, 71.
— Abbasy, M. A., and Yudkin, J. (1936) *Ibid*, **1**, 1488.
Heinemann, M. (1936) *Biochem. J.* **30**, 2229.
Hess, A. F. (1917) *J. Amer. med. Ass.* **48**, 235.
Hopkins, F. G., and Morgan, E. G. (1936) *Biochem. J.* **30**, 1446.
Johnson, S. W., and Zilva, S. S. (1934) *Ibid*, **28**, 1393.
Kellie, A. E., and Zilva, S. S. (1936) *Ibid*, **30**, 360.
King, C. G. (1936) *Physiol. Rev.* **16**, 238.
Park, E. A., Guild, H. G., Jackson, D., and Bond, M. (1935) *Arch. Dis. Childh.* **10**, 265.
Pitts, A. T. (1935) *Ibid*, p. 295.
Rhinehart, J. F. (1935) *Arch. intern. Med.* **9**, 586.
Röhmer, P., and Bezssonoff, N. (1935) *Arch. Dis. Childh.* **10**, 319.
Rusznyák, S., and Szent-Györgyi, A. (1936) *Nature, Lond.* **138**, 27.
Schultz, M. P. (1936) *J. clin. Invest.* **15**, 385.
Selleg, I., and King, C. G. (1936) *J. Nutrit.* **11**, 599.
Sendroy, J., and Schultz, M. P. (1936) *J. clin. Invest.* **15**, 369.
Sharp, P. F. (1936) *Science*, **84**, 461.
Smith, D. T., and McConkey, M. (1933) *Arch. intern. Med.* **51**, 413.
Strauss, M. R. (1936) *New Engl. J. Med.* **215**, 1164.
Taylor, S. (1937) *Lancet*, **1**, 973.
Warner, E. G., and Winterton, F. G. (1935) *Quart. J. Med.* **28**, 227.
Wilkinson, J. F., and Atkinson, C. A. (1936) *Lancet*, **2**, 967.
Wolbach, S. B. (1936) *J. Amer. med. Ass.* **108**, 7.
Wright, I. (1936) *Amer. J. med. Sci.* **192**, 719.
Zilva, S. S. (1936) *Biochem. J.* **30**, 1419.

THE EFFECT OF ŒSTROGENIC AND OTHER SEX HORMONES ON THE RESPONSE OF THE RAT TO VASOPRESSIN

By F. B. Byrom, M.D., M.R.C.P. Lond.*

(*From the Bernhard Baron Institute of Pathology, the London Hospital*)

It has been shown by several workers that lesions suggestive of eclampsia (Fauvet 1931, Byrom 1937, Végh and von Pallos 1937) and of certain other vaso-renal complications of pregnancy (Byrom 1937) can be provoked in animals by repeated subcutaneous injections of the pressor hormone of the pituitary. These lesions are evidently expressions of intense vascular spasm and the parallel lends support to the prevailing view that eclampsia is an expression of vasoconstriction. The amount of vasopressin needed to cause a morbid degree of spasm is so large, however, that it is difficult to believe that eclampsia can be due, at least wholly, to over-secretion of this hormone.

The question therefore arises whether some dislocation of equilibrium arising out of pregnancy may render the vessels hypersensitive to vasopressin. In considering this possibility a promising clue seemed to be offered by the uterine muscle, the response of which to oxytocin, a hormone closely allied to vasopressin, can be greatly enhanced by œstrin (Bourne and Burn 1928) and, on the other hand, inhibited by progestin (Knaus 1929). I therefore decided to study the effect of various sex hormones on the response of the rat to vasopressin, using renal damage as a convenient yardstick.

THE EXPERIMENTS

Except where otherwise indicated the animals used have been immature unspayed female Wistar rats, from 30 to 80 g. in weight, obtained from Messrs. Glaxo Laboratories.

After treatment with sex hormone (v. infra) the rat, together with a control of the same age, is given a single large dose of vasopressin (Pitressin, Parke, Davis and Co.). To avoid leakage the rat is anæsthetised, the injection is made through shaved skin, and the puncture mark is sealed with collodion. On the following day the rat is killed and the kidneys and other organs are inspected. Histological examination is not necessary. A convenient threshold dose of vasopressin is 50 international units per 100 g. of body-weight. In the normal rat this either leaves the kidneys undamaged or, at most, causes a few blotchy areas of ischæmic degeneration in the cortex, with or without slight injection of the intermediate zone. If this is taken as a Grade I reaction, the reaction in sensitised animals may be arbitrarily divided into two further grades of severity. In Grade II the kidney displays a row of conspicuous anæmic infarcts, with broad hæmorrhagic borders, symmetrically distributed throughout the deeper layers of the cortex (see Figure). In Grade III the response is severe enough to kill the animal within a few hours, that is before definite infarcts have had time to appear. The kidneys have a soft patternless hæmolysed cut surface, and on histological examination show extreme albuminous and

* In receipt of a full-time grant from the Medical Research Council.

dropsical degeneration and much necrosis of tubular epithelium, while the epithelial cells lining Bowman's capsule are often enlarged to the same size as those of the subjacent convoluted tubule and show similar dropsical degeneration and necrosis. Many capsules thus contain a mass of large epithelial cells which tends to compress the glomerular tufts. In Grade III, and in the more severe examples of Grade II, the bladder contains brown altered blood and the serous cavities, especially the pleuræ, are full of straw-coloured exudate. The spleen is generally shrunken and sometimes infarcted.

RESULTS

Gonadotropic hormone.—(Aqueous solution of extract of pregnancy urine,[1] doses of 10–50 rat units twice daily over periods of two to twenty-four days.) In 20 out of 25 experiments with gonadotropic hormone the subsequent response to vasopressin was greater than that of the corresponding control. Sensitisation appears after three or four days' treatment and is generally limited in degree to Grade II, doses of 50 rat units being no more effective than doses of 10 units. Sensitisation cannot be produced in ovariectomised rats (9 out of 9 experiments). It is not modified by simultaneous treatment with progesterone in daily doses of up to 1·0 mg. (5 out of 5 experiments). It is increased by simultaneous administration of œstrin[2] (5 out of 5 experiments). These findings show that gonadotropic hormone, although predominantly luteinising in action, causes sensitisation by stimulating the ovarian secretion of œstrin.

Œstrin-sensitisation of the rat's kidney.—The kidney on the right is from an infant female rat treated with large doses of œstradiol benzoate for five days, followed by a subcutaneous injection of vasopressin (50 units per 100 g. body-weight). That on the left is from a litter-mate which was given the same dose of vasopressin, but no œstradiol. The former shows confluent infarction of the cortex. The latter appears normal.

Œstrin alone.—(Aqueous solution containing 200 international units per c.cm.[3]; oily solution of œstrone, 1 mg. per c.cm.[4]; oily solution of œstradiol benzoate, 5 mg. per c.cm.[5]; doses of from 0·01 to 0·15 mg. daily over periods of five to twelve days.) In 30 out of 32 experiments œstrin has been found to sensitise the rat to vasopressin (see Figure). The degree of sensitisation bears a more direct relationship to the dose and is, in general, greater than that caused by gonadotropic hormone. With an oily solution of œstrone containing 1 mg. per c.cm. the minimal effective dose in the intact infant rat is about 0·01–0·02 mg. per day for five or more days. In the spayed infant, which is more responsive to œstrin (18 out of 18 experiments), distinct sensitisation has been evoked with as little as 0·002 mg. œstradiol benzoate (oily solution containing 0·2 mg. per c.cm.) repeated daily for twelve days.

So far the analogy with the uterine muscle is close. It remains to determine whether progesterone is capable of preventing œstrin-sensitisation. In attempting to decide this question synthetic progesterone[6] has been given, together with or preceded by œstrone, over periods of five to fifteen days. The ratio of progesterone to œstrone has been as much as 2·5 to 0·002 mg. daily. Out of 38 experiments in which the control received equal amounts of œstrone, the response to vasopressin was unchanged in 24, greater in 11, and less in only 3 instances. Where œstrone was not given to the control group the response of the group receiving œstrone and progesterone was greater than that of the control (6 out of 6 experiments).

These results are disappointing but by no means conclusive. The response of an animal to progesterone is contingent on the presence of œstrone in proportions which can only be determined by trial and error (Robson 1937). Moreover, the rat is notoriously refractory to progesterone. Even the largest doses used failed not uncommonly to suppress the œstrous character of the vaginal smear. Further research is therefore necessary before it can be concluded that progesterone has no desensitising effect.

Male hormone (testosterone proprionate) has also been considered as a possible inhibiting agent, but a few (9) preliminary experiments suggest that, on the contrary, it tends slightly to augment œstrin-sensitisation. Given alone, in daily doses of not less than 0·1 mg. it also has a slight sensitising effect, but it is much less effective, weight for weight, in this respect than either œstrone or œstradiol benzoate.

The male infant rat responds in much the same way as the female to œstrin, gonadotropic hormone, and testosterone proprionate (9 experiments), the first mentioned being by far the best sensitising agent.

Showing the Degree of Sensitisation to Vasopressin Provoked by Œstrin Treatment

Rat	Preliminary treatment.	Dose of vasopressin (units). Per 100 g. body-weight.	Dose of vasopressin (units). Actual	Grade of renal damage (v. text).
410	Nil.	60	36	Gr. I, slight.
411	Nil.	30	20	Nil.
406	Œstrogenic hormone.	15 (1/2 & 1/4 of control)	8	Gr. II, severe.
407	Œstrogenic hormone.	10 (1/3 & 1/6 „)	6·2	„ II, mod.
408	Œstrogenic hormone.	6 (1/5 & 1/10 „)	3·2	„ I, mod.
409	Œstrogenic hormone.	4·3 (1/7 & 1/14 „)	2·1	„ I, slight.

mod. = moderate.

Rats 406–409 each received five daily doses of 0·15 mg. of œstradiol benzoate and a final injection of 50 units of aqueous theelin immediately before treatment with vasopressin. The *volume* of the vasopressin injection was the same in all experiments—i.e., 3 c.cm. per 100 g. body-weight.

[1] Antuitrin S, Parke Davis. This preparation contains neither œstrogenic hormone nor vasopressin.

[2] The term œstrin is intended to cover the common œstrogenic hormones.

[3] Theelin, Parke Davis.

[4] Menformon, Organon Laboratories Ltd.

[5] Dimenformon, Organon.

[6] Progestin, Organon, oily solutions containing 5 and 25 mg. per c.cm. I am indebted to Messrs. Organon for a generous supply of the stronger solution.

Experiments with adult female rats have been necessarily limited by the cost of vasopressin, but the available data suggest that only gonadotropic hormone is capable of causing sensitisation. This is in keeping with the fact that the ovarian hormones have very little effect on the œstrus cycle of the adult rat.

Finally, the degree of sensitisation that can be achieved with œstrin has been roughly estimated by treating several members of a group of spayed infant rats with large doses of œstrin and then comparing the response to graded doses of vasopressin with that of the remaining unsensitised controls. The results, which are shown in the Table, indicate that a tenfold or greater increase in sensitivity can be achieved.

DISCUSSION

The main conclusion emerging from the above experiments is that it is possible to increase the sensitivity of the rat to vasopressin as much as tenfold by preliminary treatment with œstrogenic hormones, so that visible renal damage may be provoked in a rat weighing 50 g. by a single dose of as little as 2 units of vasopressin. Given such sensitisation, the possibility that natural secretion of vasopressin may reach toxic levels becomes proportionately more credible. Even so Hofbauer's (1918) pituitary hypothesis of eclampsia continues to rest solely on circumstantial evidence, and cannot be accepted as proven.

The conception of œstrin-sensitisation is nevertheless supported by evidence which, though not conclusive, is more direct. In the first place eclamptic toxæmia is peculiar to a period of pregnancy during which the secretion of œstrogenic hormone is excessive. Secondly, the incidence and severity of toxæmia and the output of œstrin both rise steadily during this period, are greatest at full term, and decline abruptly after parturition. Finally, it has been shown by Dieckmann and Michel (1937) and Schockaert and Lambillon (1937), working independently, that the eclamptic woman is hypersensitive to injected vasopressin.

There are therefore good grounds for suspecting that œstrin-sensitisation may be in some way concerned in the causation of eclampsia; but, if so, this sensitisation cannot be explained in terms of simple over-secretion of œstrin, for the normally pregnant woman, who secretes at least as much œstrin as the eclamptic (Smith and Smith 1935 a and b, 1937), is not hypersensitive but refractory to vasopressin (Schockaert and Lambillon). To reconcile this discrepancy with the idea of œstrin-sensitisation it seems necessary to postulate an additional inhibitory factor, which in normal pregnancy holds the sensitising effect of œstrin in check and which is lacking in eclampsia. The analogy of the uterine muscle points to progesterone, or some associated hormone, as a likely inhibitory factor. The present negative results do not exclude this possibility, and it may be recalled that Robson and Paterson (1937) and Young (1937) have independently and for quite different reasons suggested that progesterone deficiency may be concerned in eclampsia.

Unfortunately nothing is yet known about the secretion of progesterone in the later stages of either normal or toxæmic pregnancy, for it is not excreted, as such, in significant amounts. An excretion product of progesterone, pregnanediol, has however recently been recovered in considerable quantities from pregnancy urine (Venning and Browne 1937) and the progesterone deficiency hypothesis is not likely to remain long in doubt. Meanwhile the only practical inference that may be drawn from the present results is that they cast doubt on the wisdom of using œstrogenic hormones in the treatment of a condition which, for all that is known to the contrary, may be an outcome of relative over-secretion of œstrin.

SUMMARY

It is shown that the sensitivity of the rat to toxic doses of vasopressin can be greatly enhanced by preliminary treatment with œstrogenic hormone.

It is recalled that injections of vasopressin cause eclampsia-like lesions in animals, that the eclamptic patient is said to be hypersensitive to vasopressin, and that the period of pregnancy to which eclampsia is peculiar is characterised by a high output of œstrogenic hormone.

It is therefore suggested that œstrin-sensitisation may be in some way concerned in the genesis of eclampsia.

The expenses of this research have been defrayed by consecutive grants from the research funds of the London Hospital and the Medical Research Council. Generous contributions of hormones have also been made by Messrs. Parke Davis (Pitressin, Theelin, and Antuitrin S) and Messrs. Organon (Progestin, Menformon, and Dimenformon).

REFERENCES

Bourne, A. W., and Burn, J. H. (1928) *Lancet*, **2**, 1020.
Byrom, F. B. (1937) *J. Path. Bact.* **45**, 1.
Dieckmann, W. J., and Michel, H. L. (1937) *Amer. J. Obstet. Gynec.* **33**, 131.
Fauvet, E. (1931) *Klin. Wschr.* **10**, 2125.
Hofbauer, J. (1918) *Zbl. Gynäk.* **42**, 745.
Knaus, H. (1929) *Arch. Gynaek.* **138**, 201.
Robson, J. M. (1937) *J. Physiol.* **88**, 100.
— and Paterson, S. J. (1937) *Brit. med. J.* **1**, 311.
Schockaert, J. A., and Lambillon, J. (1937) *Brux. méd.* **41**, 1468.
Smith, O. W., and Smith, G. van S. (1935a) *Surg. Gynec. Obstet.* **61**, 27.
— — (1935b) *Ibid*, p. 175.
— — (1937) *Amer. J. Obstet. Gynec.* **33**, 365.
Végh, L., and von Pallos, K. (1937) *Klin. Wschr.* **44**, 1537.
Venning, E. H., and Browne, J. S. L. (1937) *Endocrinology*, **21**, 711.
Young, J. (1937) *Brit. med. J.* **1**, 953.

CAVERNOUS HÆMANGIOMA OF THE ORBIT

SUCCESSFULLY REMOVED BY KRÖNLEIN'S OPERATION

By H. B. Stallard, M.D. Camb., F.R.C.S. Eng.

ASSISTANT OPHTHALMIC SURGEON TO ST. BARTHOLOMEW'S HOSPITAL; ASSISTANT SURGEON TO THE MOORFIELDS EYE HOSPITAL; OPHTHALMIC SURGEON TO MOUNT VERNON HOSPITAL

It is rarely that an opportunity arises for the successful removal of a neoplasm from the back of the orbit by a Krönlein's operation, which leaves the patient with no gross damage to vision or to important orbital structures such as the nerves supplying the extra-ocular muscles.

CASE REPORT

A sugar-factory hand, aged 36, had noticed that his right vision had been gradually failing for 9 months. He had had no pain and was in good health. There was no history of trauma.

EXAMINATION

The right vision was reduced to counting fingers at 1 metre, the right eye was displaced upwards and forwards. The pupil was equal in size to that of the left eye, it was active to light directly and consensually, and to accommodation. The movements of the globe were normal. There was papillœdema of the upper half of the optic disc and the œdema extended into the adjacent retina. The right visual field was full and the blind spot was enlarged to an extent compatible with the papillœdema

The retinal arteries and veins were normal in size and the intra-ocular pressure was within physiological limits. The Wassermann reaction was negative; a differential blood count presented no abnormal features; and a radiogram of the orbit, accessory nasal sinuses, and skull revealed no sign of a pathological lesion. A general medical examination did not detect a primary neoplasm or other evidence of organic disease. The left vision was 6/5, the eye healthy, the visual field full, and the blind spot normal in size.

My impression was that the proptosis was due to a new formation, probably neoplastic and not inflammatory, situated within the cone formed by the recti muscles below the optic nerve and outside its sheaths, and causing mechanical compression sufficient to obstruct some of the lymphatic and venous drainage from the optic nerve and optic disc.

My reasons for believing that the neoplasm was outside the optic nerve and its sheaths were: (1) primary neoplasms of the optic nerve are slow-growing and one producing the degree of proptosis present in this patient would have caused optic atrophy and considerable visual field defects; (2) a meningioma arising from the sheath of the optic

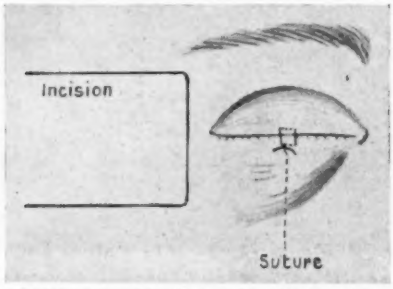

FIG. 1.—Incision for skin flap and mattress suture closing eyelids.

nerve and encircling it would on reaching the size of the neoplasm in this patient have compressed the optic nerve considerably and produced substantial loss in the peripheral visual field, and possibly disturbances in the choroidal circulation, resulting in simple retinal detachment with serous subretinal fluid and secondary glaucoma among the complications.

OPERATION

At St. Bartholomew's Hospital I performed a Krönlein's operation with the object of exploring the orbit and if possible removing the neoplasm. The lids were closed with a temporary suture, and the skin incision was made as shown in Fig. 1 and carried down to the periosteum of the external angular process, the malar bone, and the temporal fascia. Sutures were passed through the upper and lower angles of the skin flap and held up during the reflection of the flap. After this the flap was turned posteriorly over a piece of gauze, the sutures clamped to the sterile towels with Spencer Wells forceps, and the raw under-surface of the skin flap covered with gauze wrung out in warm saline. The orbital fascia and external lateral ligament were then incised close to the periosteum of the outer orbital margin and the orbital contents gently displaced medially by a flat retractor thereby exposing the lateral wall of the orbit as far as a level with the deepest part of the temporal fossa. The temporal fascia was also incised along the posterior margin of the external angular process and the frontal process of the malar bone and the temporal muscle was retracted posteriorly in order to expose the temporal aspect of the lateral wall of the orbit and the anterior part of the temporal fossa. Hæmorrhage from the skin flap and temporal muscle was checked by mosquito forceps, the larger vessels were ligatured with

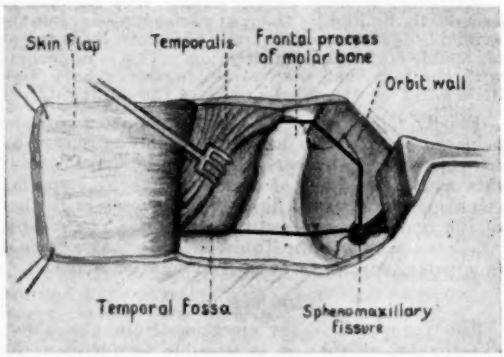

FIG. 2.—The field of operation, showing the saw cuts for the removal of a quadrilateral piece of bone from the outer wall of the orbit.

catgut, and the smaller were sealed by touching the forceps with a diathermy electrode. Retractors held the orbital contents and temporal muscle clear of the outer wall of the orbit. The periosteum was then incised and reflected for about 3 mm. over a quadrilateral shown in Fig. 2; with a small circular electrically driven saw the bone was divided along the lines of this quadrilateral, the lower cut passing posteriorly into the sphenomaxillary fissure. The outer wall of the orbit was removed and placed in normal saline at 100° F. The belly of the external rectus muscle was then divided between two mattress 000 catgut sutures. The cut ends of the muscle were reflected anteriorly and posteriorly.

The posterior part of the globe and the optic nerve were identified and the orbit was gently explored with the gloved forefinger. A neoplasm of dark purple colour, firm elastic consistence and about the size of a damson was found embracing the optic nerve below and on the nasal side. A considerable part of it was on the nasal side. Anteriorly it extended up to the sclera and posteriorly it went into the apex of the cone formed by the recti muscles. It had a thin capsule and several large vessels on its surface. The central retinal vessels could not be identified owing to orbital fat. The neoplasm was well defined and on digital examination it did not appear to be infiltrating its surrounding tissues; it was not attached to the periosteum and the medial wall of the orbit presented no gross abnormality on palpation.

By gentle dissection with the gloved forefinger the neoplasm was freed from the nasal side of the optic nerve and below. Attention was next paid to separating the neoplasm in the apex of the muscle cone, then to the

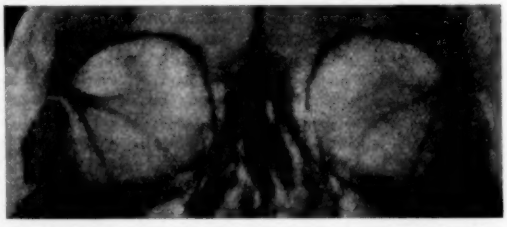

FIG. 3.—Radiogram taken one week after operation to show the position of the quadrilateral piece of bone removed from the outer wall of the right orbit.

anterior extremity and finally to the temporal side. The neoplasm was delivered by bringing it under the optic nerve and thence out of the wound. Inspection of the orbit after this procedure revealed almost the entire length of the optic nerve which appeared to be undamaged and there were no bleeding points requiring the application of pressure forceps, the cautery, or diathermy. The cut ends of the external rectus belly were sewn together with the mattress 000 catgut sutures, the quadrilateral

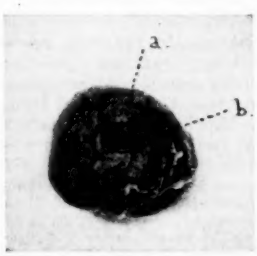

FIG. 4.—Macroscopic appearance of the upper aspect of the neoplasm. (*a*) A projection upwards on the nasal side. (*b*) Groove traversed by the optic nerve. (Natural size.)

piece of bone composing the outer wall of the orbit was replaced in position (see Fig. 3, a radiogram taken a week after operation), and the temporal muscle and orbital contents were allowed to resume their normal relationship with the outer wall of the orbit. A split rubber drainage-tube was inserted into the orbit along the angle made by the outer wall and the floor and another tube was passed subcutaneously into the lower and outer angle of the skin flap incision. The flap was carefully sewn with interrupted sutures and a firm gauze dressing applied.

PROGRESS

Three days after operation the wound was dressed and it appeared to be healing by first intention. The drainage-tubes were removed. Slight proptosis was present, the pupil was dilated, inactive to light, and slightly pear-shaped with the narrow end above. The central retinal vessels were normal in every respect. (I had feared that in removing the neoplasm some damage might have been inadvertently done to these important vessels as they could not be identified during operation.)

FIG. 5.—Photomicrograph showing some of the cavernous spaces filled with blood and lined by endothelial cells. The interstitial tissue is composed of fibrous and connective tissue, and some plain muscle-fibres are seen in the walls lining one of the cavernous spaces. (× 60.)

The papillœdema had subsided considerably and only a trace remained in the upper half of the optic disc. Abduction was limited. On the 5th day the pupil became active to light but remained dilated. A week after operation the patient began his visual improvement and examination on the 12th day showed no proptosis, good vertical movements, limited abduction, and to some extent impaired adduction; the pupil was smaller and active, only a slight trace of papillœdema remained, the right vision had recovered from counting fingers before operation to 6/12 (6/9 for 2 letters) with a +2·5 D. sph. and the peripheral visual field was full.

He was discharged from hospital and a month after operation Dr. W. A. Briggs sent me the following report :—"There is definite, though rather sluggish, contraction of the pupil to light, although, of course, it still remains pretty widely dilated. There is in addition an increasing amount of outward movement of the eye. The papillœdema has completely gone, though there are changes to be seen of a streaky nature in the region of the macula and extending to the top part of the disc. This probably accounts for the fact that his vision cannot be improved beyond 6/12 with a lens." On Oct. 28th, 1937, over five months after operation, Dr. Briggs reported that the vision was 6/9 (full), slight direct reaction of the pupil to light, very little outward movement of the right eye beyond the midline, but this was improving. Diplopia on looking to the right, but he was untroubled by this.

PATHOLOGICAL REPORT

The neoplasm measured 2·5 cm. anteroposteriorly, 2·1 cm. transversely, and 2·1 cm. vertically. It had the irregular shape seen in Fig. 4, was grooved by the optic nerve which passed over its upper surface, had a mulberry colour, was studded by small nodules, and had knots of deep purple tortuous blood-vessels traversing its thin fibrous tissue capsule. Its consistence was firm elastic, and on cutting into it the diagnosis of cavernous hæmangioma was confirmed. Fig. 5 is a photomicrograph of a section through part of the neoplasm. It is composed of widely dilated cavernous spaces filled with blood and lined by endothelial cells. Connective and fibrous tissues are present in the interstices, and a few of the vascular spaces have a coil of plain muscle in their wall suggesting a venous origin.

COMMENT

This case is of interest because it shows the possibility of removing a cavernous hæmangioma surgically without causing severe orbital hæmorrhage and without damage to such important structures as the central retinal vessels and the optic nerve which were in close relationship to it. It was evident that some damage had been done to the ciliary nerves thereby causing dilatation of the pupil and to the external rectus leading to some limitation of abduction. The latter is apparently a common complication after a Krönlein operation. These mishaps, however, are relatively trivial and apparently give rise to no unpleasant subjective symptoms. The visual recovery from counting fingers to 6/9, the full peripheral visual field, and the disappearance of proptosis have made the operation worth while.

I thank Dr. Briggs for his kindness in sending me this patient and for his valuable follow-up notes from time to time, and also Dr. H. F. Brewer for preparing the sections and microphotographs.

THE NORMAL LEVEL OF PLASMA BILIRUBIN *

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ASSISTANT IN PATHOLOGICAL CHEMISTRY AT THE SCHOOL

KNOWLEDGE of the amount of bilirubin in normal blood is at present unsatisfactory. This is in part due to the fact that many of the methods of estimating bilirubin have been unreliable and in part to the fact that no large series of values on normal healthy individuals appears to have been determined.

The object of the present investigation was, first, to determine the normal range of values in healthy

* This paper was read in the absence of the authors by Prof. Moises Chediak at the Congress of Hæmatologists, Havana, January, 1938.

individuals using a reliable method; secondly, to assess the significance of the bilirubin level in certain anæmic states in relation to the normal values found.

METHODS

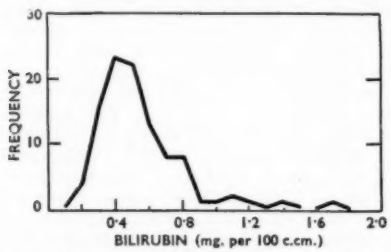
Blood was obtained by venepuncture from 50 healthy men (medical practitioners, scientific workers, and laboratory attendants) aged 16–44, and from 50 healthy women (qualified medical women and nurses) aged 20–50.

Only those samples of blood were included as normal in which the values of the red cell count and hæmoglobin estimation were within the range of normal previously indicated (Price-Jones, Vaughan, and Goddard 1935, Price-Jones et al. 1931). All samples of blood were taken between 9.30 and 10.30 A.M., the donor having had breakfast at least an hour previously.

TABLE I—PLASMA BILIRUBIN (MG./100 C.CM.)

Before breakfast.	One hour after breakfast.
1·4 .... 0·6	1·1 .... 0·5
0·5 .... 0·5	0·5 .... 0·6
1·0 .... 0·5	0·9 .... 0·6
0·5 .... 0·4	0·5 .... 0·4
1·0 .... 0·5	1·1 .... 0·4

Heparin was used throughout as an anticoagulant. Red cell counts and hæmoglobin estimations were made on venous blood by methods previously described (Price-Jones, Vaughan, and Goddard 1935). Bilirubin was estimated by the method described by Haslewood and King (1937).



Frequency distribution of plasma bilirubin in 100 healthy adults.

TABLE II—FREQUENCY DISTRIBUTION OF PLASMA BILIRUBIN

Bilirubin mg./100 c.cm.	M.	F.	Total.	Bilirubin mg./100 c.cm.	M.	F.	Total.
0·0	..	..	..	1·2	1	..	1
0·1	..	..	..	1·3	..	..	..
0·2	..	4	4	1·4	1	..	1
0·3	2	13	15	1·5	..	..	..
0·4	7	16	23	1·6	..	..	..
0·5	10	12	22	1·7	1	..	1
0·6	11	2	13	1·8	..	..	..
0·7	5	3	8	1·9	..	..	..
0·8	8	..	8	2·0	..	..	..
0·9	1	..	1				
1·0	1	..	1	Total	50	50	100
1·1	2	..	2				

M. = males; F. = females.

RESULTS

Possible effects of food intake.—In order to assess the possible effect of an ordinary breakfast consisting of bacon, eggs, toast, and coffee a series of ten estimations were made before breakfast, the last meal being dinner the night before, and an hour after breakfast. No significant difference was found (Table I). It was therefore considered justifiable to examine 100 normal subjects an hour after breakfast rather than fasting.

Normal level.—The values found in 100 healthy people for plasma bilirubin ranged between 0·2 mg. to 1·7 mg. per 100 c.cm. (Table II; and see Figure); 93 per cent. of the values were 0·2–0·8 mg./100 c.cm. All the remaining seven values were higher than 0·8 mg./100 c.cm. The high figures were all obtained in men who as far as could be ascertained were healthy and had no history of jaundice or liver disease. They had had no alcohol for the preceding 24 hours. In the two examples with highest figures estimation of bilirubin on other occasions gave figures of the same order. The values for plasma bilirubin in the 50 women cluster round the mean.

Anæmic states.—The level of plasma bilirubin found in a series of anæmic patients is shown in Table III. There were 8 cases of typical Addisonian pernicious anæmia in relapse and 1 of subacute combined system disease with megalocytic cells but an extremely mild degree of anæmia. In only 1 case was the level above the observed normal, being 2·0 mg./100 c.cm.

There were 4 cases of severe hypochromic anæmia in middle-aged women that might be described as idiopathic hypochromic anæmia; these showed figures of 0·3–0·5 mg./100 c.cm. A group of 12 cases of anæmias associated with miscellaneous conditions such as sepsis, nephritis, and hæmorrhage had values of 0·1–1·3 mg./100 c.cm.

DISCUSSION

Effect of food intake.—It has been claimed by previous observers that food intake effects the level of serum bilirubin (Perkin 1927, Barron 1931, Brøchner-Mortensen 1934–35). The level is said to be highest when fasting, protein feeding has no effect, while after carbohydrates and fat food there is a fall if a large meal is taken. The small group of results recorded here suggests that a meal of bacon, eggs, toast, butter, and coffee does not effect the level of plasma bilirubin an hour after ingestion.

Normal levels.—Previously accepted normal limits are recorded in Table IV. It is difficult to assess the value of these figures as the number of individuals investigated and their sex and age is rarely given. Individuals showing high figures were excluded in an arbitrary way by the authors as examples of "familial jaundice," a condition, however, of which no adequate description or definition has been traced. These figures cannot be excluded from the normal range without further investigation (Elton 1936, Gilbert and Lereboullet 1905, van den Bergh 1921). No evidence is given by other authors for the familial nature of the condition. The father and mother of the individual with the highest observed value in the present series had figures well within the calculated normal range. Elton (1936) claims that familial jaundice is met in approximately 3 per cent. of the white races, being especially prevalent in Jews. The bilirubin level in such individuals lies between 0·6–1·5 mg./100 c.cm. Without further knowledge of pigment metabolism in the individuals with both high and low figures it is unjustifiable to conclude that the high figures are abnormal. They may indicate, for example, a different threshold of excretion rather than of increased hæmolysis.

The following constants can be calculated from the whole series of figures obtained in the present study :—

M (arithmetic mean) = 0·539 ± 0·02467 mg./100 c.cm.
σ (standard deviation) = 0·2467 mg./100 c.cm.
ν (coefficient of variation per cent.) = 45·93 per cent.

The "skew" character of the distribution curve makes it unjustifiable to calculate the limits of normal on a basis of ±3 times the standard deviation of the mean.

It is therefore impossible with the data at present available to lay down satisfactory values for the normal limits of plasma bilirubin: 93 per cent. showed figures of 0·2–0·8 mg./100 c.cm., while the remaining 7 per cent. had values as high as 1·7 mg./100 c.cm. On the basis of these findings we would suggest that, for the present, figures between 0 and 1·3 mg./100 c.cm. should be considered as probably normal, while figures between 1·3 mg./100 c.cm. and 1·7 mg./100 c.cm. should be regarded as probably but not necessarily pathological.

TABLE III—PLASMA BILIRUBIN IN ANÆMIA

Case.	Diagnosis.	Bilirubin mg./100 c.cm.	R.B.C. mil./cm.	Hb. (per cent.).
1	Hypochromic anæmia.	0·5	4400	38
2		0·5	4800	76
3		0·3	3900	40
4		0·3	3300	36
5	Bleeding hæmorrhoids and diverticulitis.	1·3	4910	46
6	Myelo-sclerosis.	0·5	2960	49
		0·3	4200	88
		0·5		
		0·7	3700	68
7	Nephritis.	0·3	2900	54
8	Meningitis prontosil.	0·3	2800	50
9	Chronic nephritis.	0·4	1800	38
10	Uræmia.	0·5	2700	42
11	Septic leg.	0·5	3300	56
12	Arthritis.	0·3	4400	62
13	Duodenal ulcer.	0·2	3600	46
14	Pharyngeal abscess.	0·1	2410	42
15	Nephritis.	0·3	3900	62
16	Multiple injuries.	0·4	3320	54
17	Addisonian pernicious anæmia.	0·8	1700	42
18		1·0	1300	38
19		2·0	2500	58
20		1·6	1800	42
21		0·5	2840	74
22		1·0	1800	46
23 (*a*)		0·8	2030	56
23 (*b*)		0·1	1900	53
24		1·1	2100	40
25	Subacute combined system disease.	0·8	4100	92

Anæmic states.—It is often claimed that Addisonian pernicious anæmia, in contradistinction to other megalocytic hypochromic anæmias that react to liver therapy, is characterised by an abnormally high plasma bilirubin. The present figures do not substantiate this statement; 2 only of 8 patients with Addisonian anæmia showed a figure higher than 1·0 mg./100 c.cm. The group of cases is small and definite conclusions on this point cannot be drawn from the figures recorded.

The figures both for normal and abnormal bloods here recorded suggest that before any attempt is made to assess the significance of changes in the levels of serum bilirubin, studies of associated urobilin excretion should be undertaken.

TABLE IV—NORMAL BILIRUBIN

Author.	Bilirubin mg./100 c.cm. plasma or serum.	No. of examinations.
Gilbert and Herscher .. (1905)	0·25–0·357	18
*van den Bergh .. (1921)	0·08–0·24	—
Haselhorst .. (1921)	0·5	120
*Mandlebaum .. (1922)	52% above 0·2, 48% at or below 0·2	—
Hetenyi .. .. (1922)	1·21–2·00 <1·1 mg. >2·2 mg.†	85
Greene, Snell, and Walters .. (1925)	0·5 –2·0	—
Forster and Forstner .. (1926)	0·2 –1·0	—
Sivo .. .. (1927)	0·8 –1·1	—
Perkin .. .. (1927)	0·05–0·35	50
Barron .. .. (1931)	0·1 –0·5	—
Elton .. .. (1935)	0·0 –0·25	—
Tecon .. .. (1936)	0·0 –0·5	—

* The figures in these cases are given in "van den Bergh units" per 100 c.cm. and have been divided by 2·5 to convert them to mg./100 c.cm. It was pointed out by Hunter (1930) that van den Bergh had described his bilirubin standard as a dilution of 1 in 200,000, whereas it was in fact a dilution of 1 in 250,000 of bilirubin. This means, therefore, that the "van den Bergh units" must be divided by 2·5 and not by 2·0 for conversion to mg./100 c.cm. The factor 2·0 is nevertheless still found in some text-books.

† Not necessarily pathological.

CONCLUSION

The level of plasma bilirubin observed in 100 healthy adults varied from 0·2 to 1·7 mg./100 c.cm., 93 per cent. of values being below 0·8 mg./100 c.cm. The mean value for the whole series was 0·539 mg./100 c.cm. It is suggested that without knowledge of urobilin excretion in relation to plasma bilirubin it is impossible to assess the significance of these figures or those previously recorded.

We are indebted to the chief medical officer of the London County Council and to the physicians and surgeons of the British Postgraduate Medical School for permission to investigate patients in Hammersmith Hospital.

REFERENCES

Barron, E. S. G. (1931) *Medicine, Baltimore*, **10**, 77.
van den Bergh, A. A. H. (1921) *Pr. méd.* **29**, 441.
Brøchner-Mortensen, K. (1934) *Ugeskr. Laeg.* **96**, 1056.
Elton, N. W. (1935) *Ref. Gastro-enterol.* **2**, 331.
— (1936) *Ibid*, **3**, 132.
Forster, J., and Forstner, B. (1926) *Z. klin. Med.* **103**, 703.
Gilbert, A., and Herscher, M. (1905) *C.R. Soc. Biol. Paris*, **58**, 899.
— and Lereboullet, P. (1905) *Ibid*, p. 937.
Greene, C. H., Snell, A. M., and Walters, W. (1925) *Arch. intern. Med.* **36**, 248.
Haselhorst, G. (1921) *Münch. med. Wschr.* **68**, 174.
Haslewood, G. A. D., and King, E. J. (1937) *Biochem. J.* **31**, 920.
Hetenyi, G. (1922) *Z. klin. Med.* **95**, 469.
Hunter, G. (1930) *Brit. J. exp. Path.* **11**, 407.
Mandlebaum, R. (1922) *Mschr. Geburtsh. Gynäk.* **59**, 17.
Perkin, A. S. (1927) *Arch. intern. Med.* **40**, 195.
Price-Jones, C., Dill, D. B., and Wright, G. P. (1931) *J. Path. Bact.* **24**, 779.
— Vaughan, J. M., and Goddard, H. M. (1935) *Ibid*, **40**, 503.
Sivo, R. (1927) *Biochem. Z.* **189**, 159.
Tecon, R. M. (1936) *Rev. méd. Suisse rom.* **56**, 193.

THE HEART IN DIPHTHERIA *

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The prevalence of severe diphtheria in Hull during the past six years has given opportunities for the observation and study of the toxic effects seen in this disease. This paper deals particularly with the toxic myocardium—its signs and symptoms. It attempts to classify, chiefly in a clinical manner, the varying degrees of myocardial damage with a view to prognosis and management of the cases. It is based on observations from a series of 4700 cases of faucial diphtheria, which included 461 deaths, mostly toxic, and 452 non-fatal paralysed cases.

The ground is well trodden, excellent descriptions exist, and it may seem presumptuous to enter such a field armed only with fingers, stethoscope, and sphygmomanometer. But reading over the classical descriptions and watching cases in the wards, it is felt that something may still be usefully said particularly in the roughly quantitative evaluations of the cardiac signs. Is this heart condition better to-day than it was yesterday? Of what significance is this increase in the number of extrasystoles?

As bacteriological types differ, so also, and perhaps consequent thereon, clinical types differ. It is therefore unwise to regard signs observed locally as being of general application. Different rates of toxin production and fixation must produce different clinical signs, and there is a distinct impression that the toxic damage seen in Hull recently is of different type from that seen elsewhere. The Hull cases give one more the impression of heart disease, myocardial failure as such, rather than cardiac disease associated with considerable peripheral circulatory failure that had previously been noted.

The large number of toxic deaths is due chiefly to the heavy incidence of gravis strains of *Corynebacterium diphtheriæ* during this time. The majority of toxic deaths occurred within the first fifteen days of disease. In all these cases the heart is undoubtedly affected but when death occurs early, say during the first week, cardiac phenomena are not prominent and they are so overshadowed by the general poisoning that one is inclined to say that these early deaths are due to general toxæmia. Later, from the 7th to the 14th day of disease, cardiac symptoms and signs are more in evidence and the impression given is that death is due to cardiac or circulatory failure. The line between general toxic death and cardio-toxic death is arbitrary and in some cases shadowy, especially about the 7th, 8th, and 9th days, but in the majority of cases the distinction is clinically recognisable. Late cardiac deaths associated with 5th and 6th week paralyses have not been seen.

There is little to say about the acute toxic death. Cardiac signs are slight and similar to those met with in any very severe toxæmia. My chief material will be severe toxic cases which have survived the first week of illness and which either die round about the 11th day of disease or which make a slow recovery, usually complete. A few, having survived the perils of severe cardiac disease, fall victims to a late respiratory paralysis.

THE PULSE-RATE

These cardio-toxic cases, the "early heart" cases of the classical writers, show various symptoms and signs. Following a severe infection of nasopharyngeal type, with evidence of toxæmia shown by periadenitis, drowsiness, hæmorrhages, and toxic appearance, definite heart signs begin to be noticed about the 7th day. The first of these is usually in connexion with the pulse; decreasing force may be noted but the most characteristic changes are associated with rate. A high rate at first is followed by a characteristic fall. The patient may die in this bradycardiac phase but if he survives it lasts for a few days and then the rate rises again. If the pulse is charted—and it should be charted in every case of diphtheria because it gives us more information than the temperature chart—these alterations in rate give a characteristic curve. In the most severe cases one gets what might be called a Type I curve (Fig. 1). There is a considerable and rapid fall, a low bradycardiac level, and a rapid rise to a rate a little less than before the fall. In Type II both the fall and the rise are more gradual and the curve instead of following the lines of a cup as in Type I is more like that of a saucer cut in section (Fig. 2), the shallower the curve the less the significance. The heart that has escaped serious damage will not show either curve, but after an initial fall due to subsidence of fever will maintain an almost constant level.

In describing these pulse charts certain precautions are implicit. The patients are at rest in bed the whole time and the pulse is taken quietly by a competent nurse who counts for a full minute against a watch;

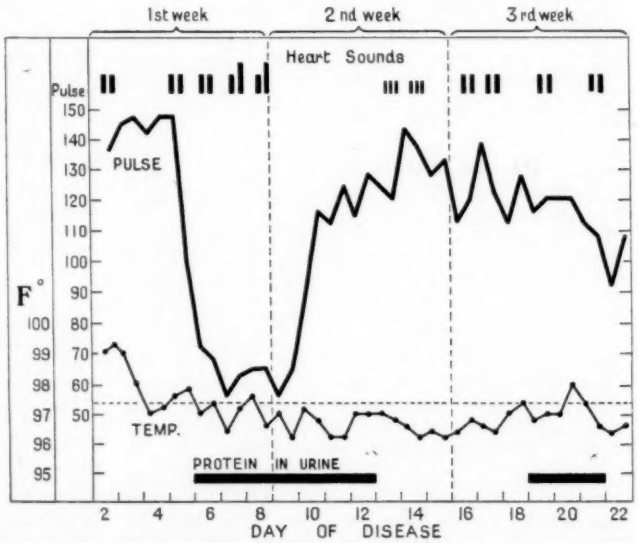

FIG. 1.—Clinical chart of girl aged 2¼, with severe diphtheria and eventual recovery, showing a Type I (cup-shaped) pulse curve and alterations in heart sounds. The first and second heart sounds are represented by columns and the lengths of these show the relative loudness; the three columns together represent a reduplicated second sound causing triple rhythm.

* A paper read to a meeting of the Fever Hospital Medical Services Group of the Society of Medical Officers of Health.

she repeats the observation if there is a considerable divergence (say more than 15 beats) from the previous reading. Subsequent pyrexia due to complications may modify the pulse curve. Pulse charts taken with these precautions are most valuable guides. An interesting feature is that these rate curves follow the blood-pressure curves. As the pulse-rate falls so does the blood pressure, and the day showing the most pronounced bradycardia is usually the day when the lowest blood-pressure readings are obtained. This, which is a reversal of the physiological rule that the lower the blood pressure falls the more rapid the pulse becomes, is both an indication and a measure of cardiac and circulatory impairment. A colleague, Dr. Robert Moyes, studied the blood pressure in 120 children at Cottingham throughout their stay in hospital and in practically all cases the blood pressure and pulse-rate curves showed correspondence. If therefore we have satisfactory pulse records we do not need the more laboriously gathered blood-pressure records; the former may be taken as a shadow of the latter (see Fig. 3).

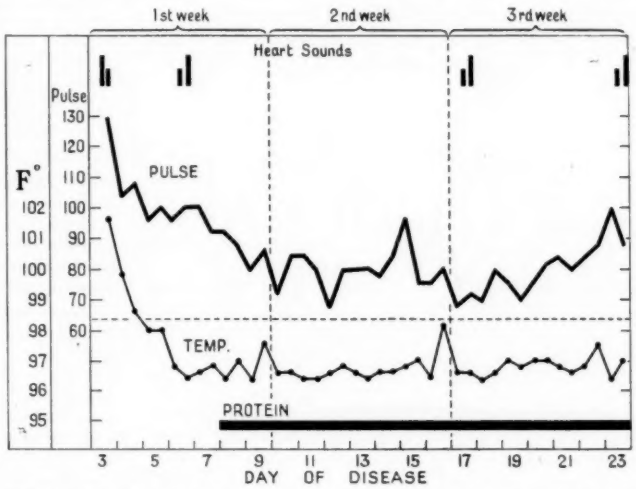

FIG. 2.—Clinical chart from boy aged 3½, with moderately severe diphtheria, showing a Type II (saucer-shaped) pulse curve and alterations in heart sounds.

Most cardio-toxic deaths take place within the bradycardiac phase. A pulse-rate of less than 50 is dangerous; of less than 40 is usually fatal. The lowest rate I have recorded is 18, felt at the wrist and heard at the apex a few hours before death. If the bradycardiac stage is passed the outlook improves with the rising rate, though a second tachycardia may be ominous if associated with other cardiac signs. A very feeble pulse, a dicrotic pulse, or a pulse imperceptible at the wrist are grave prognostic signs which need no discussion here.

THE HEART SOUNDS

If we pass from a consideration of pulse-rate curves to auscultatory signs we again can classify these in an order which indicates in the main increase or decrease in severity. Every text-book quotes the weakening of the first cardiac sound as evident in these severe cases and it is stated, and it is true, that this sound may become inaudible. Attempts have been made in the past to measure the sounds clinically and the differential stethoscope was devised for this purpose. In this instrument the sectional area of a sound-conducting channel can be varied, and in use this is decreased until the sound to be measured becomes inaudible, when the reading on a calibrated dial is made. By this means variations in the same sound from day to day may be noted or the relative intensities of the sounds in different areas compared. I have had a little experience of this instrument but discarded it for two reasons: first, I found the end-point—the point at which the sound *just* becomes inaudible—difficult to determine; and secondly, that in the diphtheritic heart some of the sounds are outside the range of the instrument—if the first sound is inaudible it obviously cannot be measured.

It appeared to be simpler and more satisfactory to compare the heart sounds at a given area with each other. The chosen area was the apex, by which is meant the visible apex pulsation, or if this is not apparent the outermost and lowest point of maximum thrust as determined first by the flat hand and then by the palpating finger. At the apex so defined if comparison is made between the relative intensity of the two sounds they may easily be classed in one of three ways. In the majority of normal hearts the first sound is greater in intensity than the second, and this is easily appreciated. In a weakening heart the first sound is weakened relatively to the second so that the intensity of the sounds approximate each other and we may say the first sound is equal to the second. Finally as myocardial weakness increases the second sound becomes relatively louder than the first. There are thus three classes, easily distinguishable after a little practice, into which we can place heart sounds at the apex: 1st > 2nd, 1st = 2nd, 2nd > 1st.

There are certain precautions to observe in using this rough clinical measurement.

Auscultation must be at the apex as defined; and it must be remembered that sometimes a normal heart may not give the usual sound ratio. In some children with apparently healthy hearts the second sound is louder than the first at the apex and in these cases the preponderance seems to be due to a relative increase of the second sound rather than to a weakening of the first. It is generally associated with an accentuated pulmonic second sound and is possibly due to abnormal sound conduction owing to anatomical peculiarities. An isolated observation, therefore, of 2nd > 1st at the apex should not be regarded as significant. It is change of these ratios in the same heart during the course of observation that is so important.

Another precaution to be borne in mind is that it is the intensity—the relative *loudness*—of the sounds we are comparing. Quality differences must be ignored. There is a tendency because of its sharp staccato quality to over-rate the intensity of the second sound.

Sometimes when differences between the sound intensities are slight, it is difficult to say which is the louder. If in doubt classify as 1st = 2nd. If it is necessary to listen for a long time then the sounds *are* approximately equal. The common normal 1st > 2nd or the abnormal 2nd > 1st are almost immediately recognisable as such.

If the sounds are equal note the length of the diastolic pause, if long these sounds are equal with normal spacing, if the pause is shortened the sounds are those usually described as tic-tac.

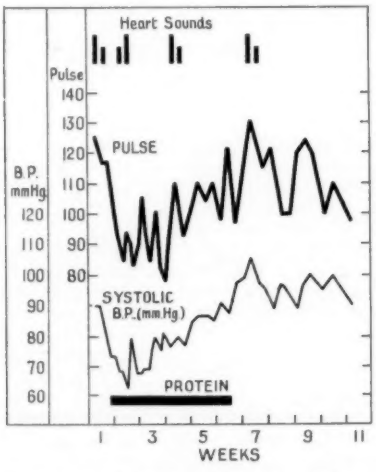

FIG. 3.—Severe diphtheria in child aged 5. Blood pressure and pulse-rate compared throughout ten weeks of illness. Note general similarity of curves, pressure rising with rate. Onset of palate paralysis in third week. Patient up in blankets in eighth week. The chart is typical of findings in severe and moderately severe diphtheria.

The heart should be examined under the conditions of absolute rest and at the same times daily. After comparing the sounds the other areas should be examined in the usual way and any abnormalities of rhythm noted, particularly the presence or absence of extrasystoles. If these are present the number per 100 beats palpable at the wrist should be noted; if the physician counts the extrasystoles by listening at the apex the nurse can count the beats at the wrist and signal when she gets to 100. If extrasystoles are less than 1 in 100 they may be described as occasional; a very usual figure is 5 or 7, but we have recorded as many as 25 or 30. Other points to listen for are splitting of the sounds, and if reduplications occur to note which sound is affected and if the reduplication is occasional, intermittent, or constant.

By using these criteria—the relative intensity of the sounds at the apex, the regularity or otherwise, the relative number of extrasystoles, and qualitative alterations in the heart sounds, particularly reduplication—we can form a scale which shows increasing or decreasing cardiac dysfunction. If a severely poisoned myocardium be observed from the day of admission the changes are fairly constant, and the average case will run somewhat as follows:—

During the first four or five days of disease the first sound is greater than the second; about the 7th or 8th day the sounds become equal, and then the second sound becomes greater than the first, then extrasystoles may appear and may increase in frequency. If the cardiac condition improves extrasystoles become fewer and disappear but the sound ratio 2nd > 1st may persist for a long time. If the condition of the heart gets worse, splitting of the second sound at the apex occurs, at first inconstant or in small groups, later constant and there is an established triple or gallop rhythm. Established gallop rhythm though of the gravest prognostic import is sometimes recovered from. In other cases the first sound is reduplicated and it would appear that triple rhythm due to a splitting of the first sound is of more serious omen than that due to reduplication of the second. Death generally occurs within a few hours.

In general therefore the auscultatory signs can be placed in a scale in the order of increasing severity somewhat as follows.

(1) Sounds, 1st > 2nd
(2) ,, 1st = 2nd } regular.
(3) ,, 2nd > 1st
(4) ,, 2nd > 1st with few extrasystoles.
(5) ,, 2nd > 1st ,, numerous extrasystoles.
(6) Intermittent reduplication of 2nd sound.
(7) Sustained reduplication of 2nd sound.
(8) Sustained reduplication of 1st sound.
(9) Inaudible 1st sound.

From any point on this scale, except possibly the last two, improvement may take place and the order of change be reversed. The scale should always be considered in conjunction with the pulse-rate curve, prognosis improving with rate except in the cases showing reduplication.

There seem to be two stages at which extrasystoles may be noted. They may occur during the second week when if numerous they indicate, in conjunction with other signs, severe myocardial damage. The second stage is from the 5th to 8th week when extrasystoles may appear and be very numerous, but their presence then is not nearly so significant and has not, in our cases, been associated with any serious cardiac phenomena.

In all these severe cases proteinuria is almost a constant accompaniment and its presence or absence should be charted regularly to assist in the general interpretation of graphical records.

PROGNOSIS

If we now combine these observations we are in a position to determine clinically the condition of the heart with a sufficient degree of accuracy to gauge progress; to give a sound prognosis; and to be a guide for general management and treatment of the case. If the statements made are substantially accurate we can say in surviving cases after three weeks' observation whether the heart is affected or not in some such manner as this.

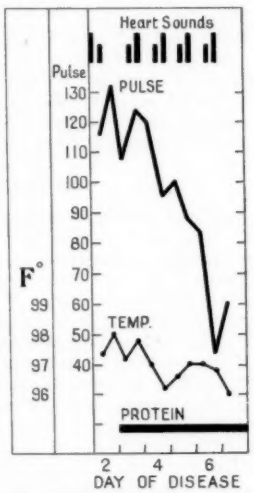

FIG. 4.—Clinical chart from a fatal case of toxic diphtheria (boy aged 12). This shows the characteristic fall of pulse-rate, and might be called the first half of a Type I pulse curve.

Myocardium not affected.—Pulse curve L-shaped or flat. Heart sounds 1st > 2nd, regular. Proteinuria absent. Such a case, even if local throat signs have been severe, has escaped toxæmia either by virtue of small amounts of natural antitoxin present in the blood or by the early intravenous administration of horse antitoxin. The patient may safely be allowed up in his fourth week and will not suffer from any late paralysis.

Myocardium slightly affected.—Pulse curve not conspicuous or shallow saucer (Type II curve as in Fig. 2). Sounds 1st = 2nd or 2nd > 1st. Proteinuria absent or limited to a few days in the second or third week. Care is needed here, rest in bed till about the sixth week and paralysis a possibility.

Myocardium severely affected.—Pulse curve deep cup (Type I as in Fig. 1). Sounds after first few days 2nd > 1st, sometimes extrasystoles. Proteinuria constant for the first two or three weeks. Prognosis depends on recovery

of rate without reduplication, but if there is heart recovery it will be followed by severe paralyses.

Myocardium fatally affected.—Pulse curve partial Type I—fall only (as in Fig. 4). Bradycardia 40 or below. Sounds after the pulse-rate falls 2nd > 1st, perhaps numerous extrasystoles; splitting of first or second sound. Inaudibility of first sound. Heavy proteinuria. Death 6th–16th day most commonly. Occasionally the fall in pulse-rate is not seen and triple rhythm is associated with a maintained tachycardia.

The most delicate sign of toxin fixation during the first three weeks is weakening of the first sound to give an apical ratio of 1st = 2nd. The next is proteinuria about the end of the third week. Often both are found together. In the absence of both there has been no severe toxæmia.

CONCLUSIONS

It is suggested that by making use of observations such as these it is possible to plan our general management of diphtheria cases more efficiently. As a rule diphtheria cases are over-treated as regards rest. This is due to excessive caution. Severe cases will certainly need 7, 8, or even 12 weeks in bed and must have this treatment. But mild and moderate cases that have not given the indications of toxæmia specified may safely be sent home at the end of their fourth week.

With regard to treatment I have little to say; rest, absolute rest, is still all-important in the cardiotoxic cases. Whether any drug influences this condition for good is open to doubt. Adrenaline was noted in some of our earlier cases to increase the number of extrasystoles with resulting decreased cardiac efficiency. I have discontinued its use entirely. When one considers the electrocardiogram and the general indication of interference with conduction the use of the digitalis group seems to be contra-indicated.

Of other drugs, camphor, Coramine, and lately Eucortone and vitamin-C concentrates one can only say that they do not alter to any appreciable degree the clinical picture.

Survival depends upon the amount and distribution of undamaged or lesser damaged myocardial tissue and it is difficult to see how the stimulation of this modicum is helpful. Conservation should be the aim; the control of vomiting in toxic cases by glucose and insulin, and the careful use of morphine to secure the all-important rest are indirect methods of cardiac treatment that mean more for the patient in the present state of our therapeutic armamentarium than any cardiac drugs.

Some of the clinical phenomena discussed may be explained simply, but the cause of others is obscure and demands the careful comparison of clinical and electrocardiographic findings. Some work has been done in this respect with Drs. J. W. Brown and D. C. Muir, and serial electrocardiograms have been taken of two groups of cases, totalling 97, in different years. At the same time a number of post-mortems on these cases have been performed and an attempt is being made to evaluate the significance of certain of the records obtained. This work will appear later, and meanwhile the investigation continues.

I should like to express my gratitude to Dr. Robert Moyes for most valuable help and the use of his records.

ROYAL EASTERN COUNTIES' INSTITUTION FOR THE MENTALLY DEFECTIVE.—On Jan. 7th the research laboratories attached to this institution at Colchester were informally opened. Dr. L. S. Penrose is directing the work.

OUTBREAK OF ACUTE RESPIRATORY INFECTION IN A NEONATAL WARD

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IN the month of March, 1937, an outbreak of respiratory infection occurred among the infants in a maternity ward of Hammersmith Hospital, 7 cases developing in the course of 31 days. The infection remained limited to one ward of the hospital, no case occurring among the 90 infants admitted to the three other lying-in wards during this time. The ward in question accommodates 18 mothers and babies, who remain in hospital for an average of twelve days after delivery. During this period 56 infants passed through the ward.

Staphylococcus aureus was obtained from the upper respiratory passages of every case that was investigated bacteriologically.

An isolated epidemic of this kind appears to be sufficiently rare to be worthy of record.

CLINICAL PICTURE

Two infants had been discharged from hospital before the onset of symptoms and were readmitted in a moribund state. No bacteriological investigations were made but the symptoms closely resembled those of the other cases.

The ages varied from ten to fifteen days, and 5 of the 7 infants were girls. All were well-developed healthy babies who had been born spontaneously at term, and in no case had cyanosis or signs of intracranial injury been present. The average weight was 7 lb. 6 oz., and all but one were breast-fed from birth. The Wassermann reaction of every mother was negative.

SYMPTOMS

Onset.—The incubation period appeared to be at least five days. One infant had been discharged from hospital five days before any symptom appeared, and another had been removed from the ward six days before it became ill. In 6 cases the onset was gradual, the infant at first appearing to have a slight cold. The 2 babies who had left the hospital had had these symptoms for three days before the mothers sought medical advice. In one case the symptoms came on acutely.

Nasal obstruction occurred in every case and was the first symptom to be noted in all but one. A mucopurulent secretion was present in 4 cases and a thin watery secretion in the remaining 3. Blocking of the nose led to difficulty in taking the breast, and a refusal to do so was often one of the first symptoms to be noted.

Cough was an early and constant symptom and was only absent in one case where the infection remained limited to the nasal passages. As the illness progressed the cough occurred in frequent paroxysms, causing considerable cyanosis in the severer cases.

Pyrexia was present in every case and was noted on the first day of the illness. High fever did not occur, the highest temperature recorded being 102·5°F. The duration varied from one to nine days, the average being five. It ran an irregular course and subsided by lysis.

Cyanosis was present in 6 cases. In the 2 fatal cases it was extreme, the infants being almost "black" when admitted. In a third case a dusky colour of the face was present for three days and then only occurred after coughing. In the remaining three cases cyanosis was not a marked feature and was only noted after spasms of coughing.

Dyspnœa was severe in three cases and less so in two others. There was Cheyne-Stokes breathing in the 2 fatal cases.

Sputum.—A striking feature in three cases was the continuous expulsion, through nose and mouth, of a copious white frothy secretion. The cases presenting this symptom were all severe and 2 died. The condition was also present, to a less extent, in a milder case.

Signs in the chest.—The 2 fatal cases had signs of massive bilateral consolidation, which was confirmed post mortem in one of them. Cases 3 and 4 showed the signs of capillary bronchitis, fine râles being heard posteriorly, especially at the bases. In Cases 5 and 6 a coarser bronchitis was present, and Case 7 had no physical signs in the chest.

BACTERIOLOGY

No bacteriological examinations were made in Cases 1 and 2. The Table shows the findings in the other cases.

BACTERIOLOGICAL FINDINGS

Case.	Throat.	Nose.
3	*Staphylococcus aureus.*	
4	*S. aureus;* pneumococcus Type 3.	
5	*S. aureus;* non-hæmolytic streptococcus.	*S. aureus;* very heavy growth.
6	Ditto.	*S. aureus:* pneumococcus Group 4.
7	Non-hæmolytic streptococcus.	*S. aureus.*

Swabs were taken from the nose and from the back of the pharynx of 5 infants with the following results. A heavy growth of *S. aureus* was obtained from the nose in every case, and from the throat in every case but one. A pure growth was obtained from the nose in three cases and from the throat in one. In the remaining cultures a mixed growth was obtained, the other organism being a streptococcus in 3 cases and a pneumococcus in 2.

SEVERITY OF ILLNESS

The earliest cases were the most severe, the first two being fatal. All the remaining infants recovered. The symptoms gradually became less severe as the later cases developed, and in the last case the condition remained localised to the nasal passages. The infection subsided spontaneously 31 days after the occurrence of the first case.

The duration of the illness varied from 2 to 9 days, the average being 6 days. Death occurred on the fourth and fifth days in the two fatal cases.

TREATMENT

Prophylactic.—Certain routine procedures are carried out in the department. Any mother suffering from a cold wears a mask while feeding the baby. Mothers and babies who develop pyrexia or signs of an infective lesion are sent to the isolation department. Nurses suffering from colds or sore-throats are forbidden to attend to maternity cases until a swab from throat and nose has been examined bacteriologically. These precautions were taken as usual, and in addition the ward was closed to further admissions as soon as it was realised that the cases of infection among the infants were related to each other. All staphylococcal carriers were removed from the ward. Infants were strictly isolated upon the first sign of a cold developing.

Curative.—The child was placed in the position in which it appeared to be most comfortable—usually slightly propped up—and was kept completely at rest, not being removed from the cot for any purpose. The mother's milk was expressed and given to the infant by bottle or by spoon, according to which method caused least disturbance. Extra fluid was administered by mouth between feeds in the form of hypotonic saline. The nasal passages were kept cleared of mucus by means of a soft rubber catheter, lubricated with sterile paraffin, which was passed as often as necessary. Oxygen was given continuously by nasal catheter to all cases showing any sign of cyanosis or dyspnœa and was helpful in lessening these conditions. Where there was much secretion of mucus the infant was given a mixture containing tincture of belladonna (℥2½ every four hours); this had a beneficial effect in *two* cases. Stimulants in the form of Coramine and brandy were given to the severe cases when necessary.

INVESTIGATION OF CAUSES

Cases of staphylococcal pneumonia in adults have been found to follow influenza epidemics (Fraenkel 1904, Chickering and Park 1919, Macgregor 1936). A severe influenza epidemic occurred in London at the beginning of this year.

Swabs from the throat and nose of all members of the ward staff and from any patient who had a cold were submitted to bacteriological examination with the following results.

A growth of *S. aureus* and pneumococcus type 3 was obtained from two nurses, and a growth of *S. aureus* alone was obtained from two other nurses and one patient. The mother of Case 6 developed acute bronchitis during the puerperium, and pneumococcus Group 4 was isolated from the sputum. This organism was also present, together with *S. aureus*, in the nasal passages of her child. The mother of Case 5 developed a mild inflammation of the left breast on the ninth day of her puerperium—two days before the onset of symptoms in her child—and *S. aureus* was obtained from the milk. Johnson and Meyer (1925) and Macgregor (1936) consider that aspiration of infected milk may be a cause of pneumonia in infancy. Case 3 developed a staphylococcal follicular eruption of the face and neck a few days before the onset of respiratory symptoms, and the possibility of a blood-borne infection must be considered here.

DISCUSSION

Pneumonia in the neonatal period is described as coming on within a few hours or days of birth and running a rapid course, with little in the way of symptoms and signs. Among its predisposing causes may be prematurity, asphyxia at birth, atelectasis, intracranial injury, or congenital syphilis (Browne 1922, Cruickshank 1930, Warwick 1934, Craig 1935). Browne and Craig consider that aspiration of infected liquor amnii may be a cause of infection.

The outbreak described differs from this type of case. The infants were a little older, and the healthy babies in the ward were affected, whereas the delicate ones were not. A definite train of symptoms occurred, all the cases running a similar course.

Macgregor (1936) describes 10 cases of staphylococcal pneumonia in children, in which similar symptoms were present. Fraenkel (1904) draws attention to the profuse

frothy sputum which frequently accompanies the condition in adults. Smith (1935) gives an account of 4 cases of pneumonia of staphylococcal origin, occurring among the infants in a maternity ward. The symptoms resembled those of the cases already referred to, but were of greater severity, all 4 cases being fatal.

Infants in the neonatal period appear to be especially susceptible to staphylococcal infections. Von Reuss (1921) states that pemphigus simplex is almost always due to this organism, and that conjunctivitis, omphalitis, and other lesions are frequently caused by it. Graham (1925) describes two cases of staphylococcal pyuria in new-born infants. Cases of enteritis at an early age have been attributed to this organism (Schabort 1906, Kermauner and Orth 1905). It is possible that the staphylococcus plays a more important part in the production of lesions of the respiratory tract in infancy than has hitherto been supposed.

Although *S. aureus* is occasionally isolated, it does not appear to be a normal inhabitant of the upper respiratory passages of the healthy infant. Swabs were taken from the nasal passages of 10 infants in another ward of the hospital, whose ages varied between two and thirteen days; 7 yielded *Staphylococcus albus*, one a mixed growth of *S. albus* and *aureus*, and two a small growth of *S. aureus*. These findings are consistent with those of Smith (1935) who, in an investigation of 16 healthy infants, obtained a scanty growth of *S. aureus* from the upper respiratory passages of 6, whereas in cultures taken from infants who were contacts of known cases of staphylococcal pneumonia, a heavy growth of this organism was obtained in 12 out of 15 cases.

The staphylococcus may have been the primary invader in the cases described, or it may have been secondary to some other organism. It was, however, the only organism that was constantly present. The source of infection is uncertain, but several staphylococcal carriers were found among nurses and patients.

SUMMARY

1. An isolated outbreak of 7 cases of respiratory infection in the neonatal period is described.
2. *Staphylococcus aureus* appeared to be the causal organism.
3. The ages of the infants varied from 10–15 days and all had been healthy at birth.
4. The incubation period was at least 5 days.
5. The chief symptoms were gradual onset, nasal obstruction, cough, fever, cyanosis, dyspnœa, and profuse frothy sputum.
6. The early cases were the most severe and the epidemic died out in 31 days.
7. Several possible sources of infection are considered.

My thanks are due to Sir Frederick Menzies, chief medical officer of health for London, and Prof. James Young, of the department of obstetrics, British Postgraduate Medical School, for permission to publish these cases, and to Dr. Alan Moncrieff and Dr. J. G. Scadding for help and advice.

REFERENCES

Browne, F. J. (1922) *Brit. med. J.* **2**, 590.
Chickering, H. T., and Park, J. H. (1919) *J. Amer. med. Ass.* **72**, 617.
Craig, W. S. (1935) *Edinb. med. J.* **42**, 599.
Cruickshank, J. N. (1930) *Spec. Rep. Ser. med. Res. Coun., Lond.* No. 145.
Graham, R. H. (1925) *Amer. J. med. Sci.* **170**, 401.
Johnson, W. C., and Meyer, J. R. (1925) *Amer. J. Obstet. Gynec.* **9**, 151.
Kermauner and Orth (1905) *Z. Heilk.* **26**, 194.
Macgregor, A. R. (1936) *Arch. Dis. Childh.* **2**, 64.
Schabort (1906) *Geb. u. Gynec.* **24**, 29.
Smith, C. M. (1935) *Lancet*, **1**, 1204.
Von Reuss, A. R. (1921) Diseases of the Newborn, London.
Warwick, M. (1934) *Amer. J. med. Sci.* **187**, 253.

CLINICAL AND LABORATORY NOTES

APPLICATION OF PRESSURE IN THE INJECTION TREATMENT OF HÆMORRHOIDS AND PROLAPSUS ANI

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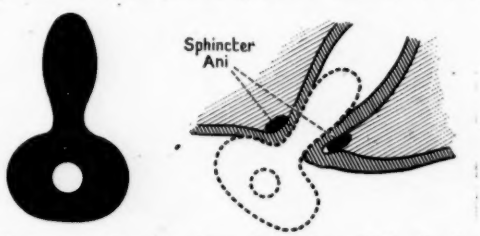
THE amount of annoyance and distress caused by hæmorrhoids needs no emphasis from me. Their treatment by injection has without doubt been a great success, and has saved many sufferers from operation and invalidism. I wish, however, to suggest a method by which its results can be further improved.

Injection therapy, as we all know, works by sclerosis of the surrounding tissues and hæmorrhoidal sinuses, and in my experience it has been possible to enhance this effect by applying pressure over the sclerosing pile. For this purpose I use a wooden plug, made from a piece of hard wood and shaped as shown in the accompanying figure. The shape ensures that on insertion into the rectum it remains in situ, while the neck (which can be increased in diameter by a layer of gauze) comes in contact with the pile-bearing area inside the anal sphincter. This area is only a few millimetres away from the sphincter ani, which presses this pile-bearing area against the hard neck of the plug. Thus we have a pressure-removal of blood from the hæmorrhoidal area, and in fact, by the continuous action of the anal sphincter, a pressure sclerosis of the hæmorrhoids and hæmorrhoidal sinuses. We can increase the diameter of the neck of this plug to suit the tone and contraction of sphincter ani, and (what is more) we can impregnate the mucous membrane and hæmorrhoidal area by applying medicaments to the gauze round the neck of the anal plug, and thus get continuous surface therapy for as long as we wish. How different this is to the old-fashioned application of unguenta to the anus, where it only reached a part of the pile, and to the many types of suppositories, which disappear in a few minutes because of their low melting-point.

The method described thus permits of continuous application of a therapeutic agent, associated with a physical obliteration and pressure trauma to the hæmorrhoids and sinuses. Medicaments on gauze can also be applied to areas of the rectum two inches above the anal sphincter and I have found the results encouraging. The insertion of this plug with a little vaseline is painless, even after injection of the pile-bearing area.



Diagrams to show the shape of the plug and its situation when inserted.

I have used the procedure described in very many cases of hæmorrhoids, whether prolapsed or not, with excellent results. A similar plug is even more useful in the treatment of prolapsus ani after the injection of the submucous tissues; but in these cases it must be a longer one. The levatores ani and sphincter ani combine to compress the tissues against the hard wood of the plug, which at the same time keeps the prolapsed tissues of the rectum in place until the formation of fibrous tissue holds them where nature meant them to stay. The plug in these cases is kept in position by a bandage between the legs, and the bandage is threaded through a hole in the head of the plug, as seen in the Figure. The patient is in bed, not ambulant like the patient with hæmorrhoids.

After injection of piles I advise that the plug should be kept inserted for five days, being removed only for evacuation of the bowels. For the next fortnight it is left in only at night. In cases of prolapsus ani in young children and old women rest in bed must be enforced, and the plug removed only for evacuation of the bowels. I make these patients stay in bed at least three weeks.

PNEUMO-ŒSOPHAGEAL FISTULA

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PNEUMO-ŒSOPHAGEAL fistula is uncommon. The case here reported is of especial interest because of the unusual type and probable site of origin of the new growth which determined the fistula.

Most of the cases on record have been in working men between the ages of 50 and 72, and the duration of life was about six months from the onset of symptoms. Gangrene of the lung was usually an early complication, but Steiner (1927) reports a case in which there was a pneumonia at the right base with no signs of gangrene for six months, although the œsophagus was perforated.

Our patient was a man of 39, a gardener, admitted to the Royal Free Hospital with pain in the right side of the chest. This had come on suddenly six months before admission, and a month later he had "belched" up blood, which gave temporary relief of pain. Three weeks before admission he had another attack of pain in the same side of the chest at the base, which was aggravated by breathing. He had a hacking cough. During this period the sputum was yellowish and extremely offensive, and he gradually developed dysphagia. From the time of onset of symptoms to the time of admission (i.e., six months) he had lost 1 st. in weight and was breathless, especially on exertion.

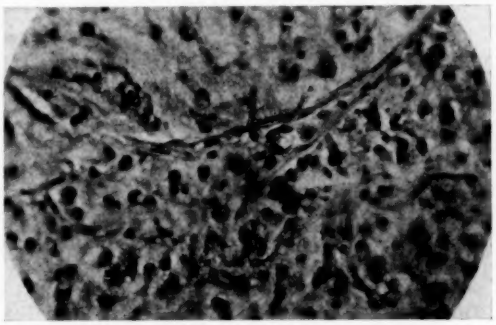

FIG. 3.—Diffuse mucoid carcinoma, showing signet-ring cells. Hæmatoxylin and mucicarmine. (× 200.)

Examination.—He was thin, sweated profusely, and had a yellowish skin, with a malar flush; there was advanced clubbing of fingers and toes, and nodules the size of a pea and of rubbery consistency were palpable under the skin of the anterior abdominal wall and over the right pectoralis major. No glands could be found in the anterior or posterior triangles. There was extreme dyspnœa and the appearance was toxæmic. Movement of the chest was diminished at the right base both anteriorly and posteriorly. Percussion was stony dull at the right base from the fourth rib down anteriorly and the sixth rib down posteriorly. Air entry was absent in this area, with decreased vocal fremitus. Ægophony was heard in the right posterior axillary line. The apex-beat was not displaced; the heart was not enlarged and there were no abnormal sounds. The sputum was offensive and contained particles of undigested food. On swallowing 2 oz. of dilute methylene-blue solution it was immediately coughed up with the sputum. Radiograms are shown in Figs. 1 and 2.

1 2

FIG. 1.—Six days before admission to hospital a barium-swallow was given and on radioscopy the fluid was seen to pass down the œsophagus into the stomach. The lower third of the œsophagus appeared constricted. A sinus at the junction of the middle and lower thirds of the œsophagus was seen to pass from the œsophagus into a cavity in the right lobe of the lung.

FIG. 2.—Radiogram taken on admission (9 days before death). The right lower lobe of the lung was converted into a large abscess cavity and contained a fluid level. It is interesting that the time interval between the two radiograms was 6 days.

Course.—Owing to the toxæmic condition of the patient and the nature of the fistula, a preliminary gastrostomy was performed with a view to drainage of the abscess later. At the operation one of the nodules was removed from the anterior abdominal wall for biopsy. It consisted of malignant epithelial cells containing mucus, the appear-

ance being strongly suggestive of primary gastric carcinoma. The gastrostomy proved unsuccessful, and the patient died six days later.

Post-mortem examination.—The lower lobe of the right lung was converted to a ragged-walled abscess cavity

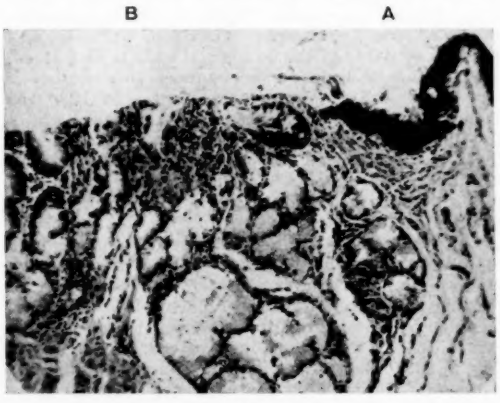

FIG. 4.—Upper cardiac glands: (A) squamous epithelium of œsophagus; (B) patch of gastric glands. Hæmatoxylin and eosin. (× 75.)

traversed by bronchi and blood-vessels and containing acid-smelling brown fluid. A large gelatinous ulcerated growth 12 cm. long occupied the whole circumference of the lower part of the œsophagus, the lower limit being 2·5 cm. above the cardiac orifice. It had begun to invade the roots of both lungs and communicated with the abscess cavity through a ragged opening in the œsophageal growth. In the upper part of the œsophagus at the level of the cricoid cartilage, on each side of the middle line anteriorly, was a linear reddish area of velvety appearance, the larger being nearly 1 cm. long. These proved to be heteroplasic patches of gastric mucous membrane.

The œsophageal, mediastinal, bronchial, cœliac, and mesenteric lymph glands were enlarged with secondary growth. Nodules of growth were present in both kidneys and both adrenals, and in the subcutaneous tissue of the anterior abdominal wall. The other organs showed little pathological change. Histologically the mucosa and whole thickness of the œsophageal wall was infiltrated by signet-ring cells, their nucleus being displaced to one side by mucus. In many areas the tumour was undergoing mucoid degeneration into a gelatinous mass (Fig. 3). The lower end of the growth was adenocarcinomatous. The secondary growths were polymorphic, containing varying amounts of mucin. The velvety patches, also the mucosa adjacent to the growth, consisted of gastric mucous membrane with glands of the cardiac and fundal type possessing chief and oxyntic cells. The limits of the patches were sharply defined from the adjacent mucous membrane (Fig 4).

The histological type of carcinoma in this case, taken in conjunction with the patch of gastric mucous membrane at the lower edge of the tumour and the patches of gastric mucous membrane in the upper part of the œsophagus, suggests that the primary growth arose in one of these heteroplasias.

In a paper on the heteromorphoses in the human body, Nicholson (1922) reviews the principal heteroplasias. He quotes Schaffer as the first to demonstrate that in about 70 per cent. of all bodies patches of gastric mucous membrane are found in the upper part of the œsophagus. These heteroplasias are termed "upper cardiac glands" to distinguish them from the lower cardiac glands which are scattered in the lower part of the wall of the œsophagus close to its opening into the stomach. Hurst and Stewart (1929) state that heterotopic gastric mucous membrane may be responsible for the initiation of peptic ulceration, and in at least three of the published cases (Fraenkel 1899, Tileston 1906, and Stewart and Hartfall 1929; all quoted by Hurst and Stewart) this association was demonstrated.

Although it is recognised that carcinoma of the œsophagus may arise in one of these heterotopic areas, few cases have been reported. Kaufmann (1929) states that "cylindrical-cell carcinomas from gland epithelium, or possibly from heterotopic gastric glands, sometimes mucoid in structure, are rare." In an analysis of 41 autopsies of carcinoma of the œsophagus, Clayton (1928) describes only 1 case, in the upper third, of an adenocarcinoma (medullary type, and no mucus-formation). He states that it arose from mucous glands in the submucosa.

Two cases (Cabot, case records, 1933, 1935) are described as adenoacanthoma. One was an extensive tumour in the lower third of the œsophagus and microscopically had the appearance of an adenocarcinoma with a little cornification. In the other case the growth was situated at the cardia and most of it was within the stomach.

We are indebted to Dr. L. S. T. Burrell for permission to publish this case.

REFERENCES

Cabot, case records (1933) *New Engl. J. Med.* **208**, 652.
— (1935) *Ibid*, **213**, 1311.
Clayton, E. S. (1928) *Surg. Gynec. Obstet.* **46**, 52.
Hurst, A. F., and Stewart, M. S. (1929) Gastric and Duodenal Ulcer, London.
Kaufmann, E. (1929) Pathology for Students and Practitioners, Philadelphia.
Nicholson, G. W. (1922) *Guy's Hosp. Rep.* January, p. 84.
Steiner, P. (1927) *Wien. med. Wschr.* **77**, 810.

MEDICAL SOCIETIES

SOCIETY FOR THE STUDY OF INEBRIETY

AT a meeting of this society on Jan. 11th papers on official contacts with drug addiction were read by Dr. W. NORWOOD EAST, H.M. Commissioner of Prisons, and Major W. H. COLES, H.M. Chief Inspector under the Dangerous Drugs Act and representative of the United Kingdom on the League of Nations Opium Advisory Committee.

Drug Addiction in Britain

Dr. NORWOOD EAST began by speaking of the legislation that discourages alcoholic addiction in this country. Among the fruits of legislative action he included the State management of licensed premises at Carlisle, which owed its inception to a decision of the Central Control Board (Liquor Traffic) in 1916. The Royal Commission on Licensing reported in 1932 that the Carlisle system was likely to reduce excess while meeting the requirements of the moderate consumer and that given the right type of management it was possible to reconcile, both in intention and practice, social benefit with good business methods. During 1936 there were 47 convictions for drunkenness in Carlisle city, or 7·96 per 10,000 population, the corresponding figures for all English county boroughs being 13·07 per 10,000. The accounts for

the year ended last March show a profit of £74,000 on a capital outlay of about £900,000. The average number of persons dealt with summarily for drunkenness, &c., in this country in the years 1910–14 was 193,354. The figure fell to 33,858 in 1932, after which there was a gradual rise to 48,110 in 1935, the last year for which figures are available. The number of persons received into prison on conviction for drunkenness was 52,149 in 1913, 5836 in 1932, and 6880 in 1935.

Turning to the arrangements made to control the manufacture, possession, and distribution of narcotic drugs, Dr. East said that the channels through which these drugs circulate legitimately are open to frequent inspection, and the addict or would-be trafficker has little chance of obtaining them from such sources without the fact of excessive supplies coming to light. There is said to be no evidence of any organised illicit traffic in drugs in the United Kingdom. The estimated population of the United Kingdom at mid-year 1936 was 47,084,753, and the approximate number of addicts known to the central office at the end of the year 1936 was 616 (313 men and 303 women). Of this total some 137 were doctors, 5 pharmacists, 3 dentists, and 2 veterinary surgeons. The drugs involved were morphine and preparations 543, heroin 61, cocaine 52, and combined drugs 50. During the year 1936 9 doctors, 1 dentist, 1 pharmacist, and 1 veterinary surgeon were convicted of offences against the Dangerous Drugs Acts.

The curative value of imprisonment for drunkenness has probably never been convincing said Dr. East. At the same time there can be no doubt that alcoholists derive benefit from even short periods of enforced abstinence. Prison medical practice has necessarily provided a large-scale experiment concerning the abrupt withdrawal of alcohol in determining the onset of delirium tremens. When cases were more numerous it was the general experience of prison medical officers that where prisoners were received verging on an attack of delirium tremens the enforced abstinence and medical care and treatment, so far from precipitating an attack, often shortened it. There is no reason to believe that this view is altered by more recent experience. In cases of addiction to narcotic drugs also, abrupt withdrawal is the practice usually adopted by prison medical officers who are in the fortunate position of having their patients entirely under control. The fact that the patient realises this and must face up to the situation has therapeutic value, and there is some reason to think that the sense of inferiority and dependence in the addict is less pronounced in the prison hospital than when at liberty, because he realises that other patients in the same hospital are often also social failures who must readjust themselves to social requirements.

International Traffic in Drugs

Major COLES said that thanks to the stricter and more general application of the international conventions there has been during the last few years a progressive shrinkage in the difference between the quantity of narcotic drugs manufactured and the quantity used for medical and scientific purposes. The statistics furnished by governments of the amounts lawfully manufactured show that these amounts approximate more and more nearly to the world's legitimate consumption, thus leaving little apparent margin available for the illicit market. It can now be said that practically no drugs that are licitly manufactured ever escape into the illicit traffic. In 1929 the known world manufacture of morphine for all purposes amounted to approximately 60 tons and the world's legitimate needs to between 31 and 32 tons, but since 1931 manufacture has approximated to legitimate requirements. There is no precise knowledge, however, of the amount consumed for maintaining addiction, nor can any accurate estimate of the number of addicts be formed. It is certain that in some countries the number is very small and in others very large. In 1930 the Canadian authorities estimated approximately 1 addict per 1000 population. The United States of America about the same time reckoned a similar figure. In 1928 it was considered that the proportion of addicts in the large towns in Germany was 1 in 10,000 and 0·56 per 10,000 elsewhere. In Egypt about 1930 it was estimated that addicts numbered 500,000 in a population of about 14,000,000. This initial figure has been much reduced in later estimates, and in 1934 the Egyptian authorities reported 18,523 addicts. In reply to a questionnaire addressed to the Chinese authorities by the League of Nations, it was stated that the number of registered opium addicts was 3,700,000. It has been stated that in Harbin there are not less than 300 heroin dens besides 100 authorised opium saloons. No one knows how many clandestine factories there are nor what is their output.

The next step in control, and it is hoped the ultimate elimination of illicit traffic, is limitation of the production of the raw materials to the quantity necessary to supply the medical needs of the world.

THE LANCET 100 YEARS AGO

January 13th, 1838, *p.* 561.

From a leading article on the influence of marriage on the duration of human life.

VOLTAIRE once asserted that the majority of persons who commit suicide are unmarried, and adduced this circumstance as a proof that the wedded state is favourable to the prolongation of life. HUFELAND, also, has established the influence of celibacy in rendering still more brief our sojourn upon this earth. . . .

ODIER has determined the mean duration of life in females from observations made during the years 1761 to 1813. . . . The difference of life, between married and unmarried females is, on an average, five years; or, to place the fact in a stronger point of view, a young woman of twenty years of age, by marrying, adds nine years and a half to the probable duration of her life. . . . Let us turn to the male sex, and for our personal comfort and satisfaction, show that, in giving life to others, the male also increases the probable duration of his own existence. . . . The facts . . . are clearly demonstrated by . . . tables which Dr. CASPAR has calculated from the documents furnished by DEPARTIEUX. . . . Between 30 and 45 years, a period during which men generally marry, we . . . find a difference of mortality which is considerably in favour of those who have taken unto themselves wives; after the forty-fifth year, this proportion on the side of married men goes on increasing; for the tables show us that, taking 100 married and 100 unmarried individuals, the number of those who live beyond the age of 45 is greater by 36·8 in the former class than in the latter . . . at a later period its influence becomes still more manifest, since for 11 bachelors who live beyond the age of 70 we find no less than 27 married men.

The facts . . . confirm . . . that the fulfilment of a pleasing duty, on the part of both sexes, is calculated to add many years to the probable duration of human existence.

REVIEWS AND NOTICES OF BOOKS

British Encyclopædia of Medical Practice

Vol. VI. By Various Authors. Under the general editorship of Sir HUMPHRY ROLLESTON, Bart., G.C.V.O., K.C.B., M.D., D.Sc., Emeritus Professor of Physic, Cambridge. With the assistance of Prof. F. R. FRASER, M.D., F.R.C.P.; Prof. G. GREY TURNER, M.S., F.R.C.S.; Prof. JAMES YOUNG, D.S.O., M.D., F.R.C.S.E.; Sir LEONARD ROGERS, K.C.S.I., F.R.C.P., F.R.C.S., F.R.S.; and F. M. R. WALSHE, O.B.E., M.D., D.Sc., F.R.C.P. London: Butterworth and Co. 1937. Pp. 602. 35*s*.

THIS volume opens with an article on gonorrhœa by L. W. Harrison and ends with one on hydrotherapy by M. B. Ray. Venereology is further represented by an article by R. V. Rajam on ulcerative granuloma and physiotherapy by a contribution by E. P. Cumberbatch on radiant heat. E. A. Cockayne's article on heredity and constitution is the sole pædiatric representative in the volume which contains in all 39 articles. Of these, 22 belong to the sphere of general medicine. Three only deal with surgical topics; Norman Lake's on diseases and deformities of the hand, G. Grey Turner's on hernia, and Harold R. Drew's on hydatid disease. There are two dermatological articles; one by Arthur Whitfield on abnormalities and diseases of the hair follicles, and one on herpes by G. H. Percival. Frank Marsh discusses heat-stroke and heat-exhaustion, conditions almost remote enough from an English winter to be fairly classified, with W. G. Liston's article on guinea-worm disease and N. Hamilton Fairley's on histoplasmosis as tropical diseases. Neurology claims four articles; on hemiatrophy and hemihypertrophy by Denis Brinton, on hemiplegia by E. A. Blake Pritchard, on hydrocephalus by C. P. Symonds, and on hepato-lenticular degeneration jointly by F. M. R. Walshe and J. G. Greenfield. Of the remaining articles, 10 deal with various aspects of cardiology. D. Evan Bedford and J. W. Brown collaborate on the subject of congenital heart disease, Reginald Miller writes on rheumatic heart disease in children, and K. Shirley Smith on pericardial diseases. A. G. Gibson contributes articles on myocardial diseases and non-malignant endocarditis. Arthur W. Falconer writes on malignant endocarditis, T. F. Cotton on mitral valve diseases, Maurice Campbell on aortic valve diseases, B. T. Parsons-Smith on diseases of the right heart, and Crighton Bramwell on heart failure. The reader may feel that an undue degree of specialisation has been shown here in the parcelling out of the heart among so many authors, any one of whom could probably have coped satisfactorily with the whole subject. The rest of the volume covers ground much of which will be familiar, some less so and some but rarely explored by the general practitioner. In the first class we have D. W. Carmalt-Jones's article on hæmatemesis, Jenner Hoskin's on hæmoptysis, J. F. Gaskell's on hæmaturia, and C. P. Symonds's on headache. To the second class belong C. W. Buckley's article on gout, perhaps G. E. Beaumont's on hæmoglobinuria, and the article on Hodgkin's disease in which the general editor collaborates with M. H. Gordon and A. E. Gow. In the third group we would place the article on hæmatoporphyrinuria by E. C. Dodds and J. Douglas Robertson, hæmochromatosis by J. H. Sheldon, hæmophilia by L. S. P. Davidson, hæmorrhagic diseases by H. Letheby Tidy, and hæmothorax by F. G. Chandler. The exacting standard set by the previous volumes in this series is well maintained in this one.

Malaria

A Handbook of Treatment. Parasitology and Prevention. By BERNARD NOCHT, M.D., and MARTIN MAYER, M.D., Professors at the Institute of Tropical Medicine, Hamburg. London: John Bale Medical Publications. 1937. Pp. 196. 10*s*. 6*d*.

Two professors of the School of Tropical Medicine in Hamburg have succeeded in condensing an enormous mass of epidemiological and other data into handy compass. This alone is a feat, and the experience and distinction of the authors lend such authority to their statements that their book is likely to take a high place among works of this kind. The clinical descriptions of the more severe forms of subtertian malaria and blackwater fever are especially useful, as is also the judicious appraisement of the action of the newer drugs—atebrin and plasmoquine—in the various phases of the disease. Quinine therapy is given due prominence. The epidemiological and laboratory sections are adequate, and the section devoted to immunity in malaria is clear and satisfactory as far as our knowledge goes. At the end of the volume there are two coloured plates depicting malaria parasites in thin and thick film preparations. *Plasmodium ovale* is not featured. This is a comprehensive and up-to-date survey of malaria and many workers in tropical medicine will be glad that it is now available in English.

Fundamentals of Anatomy

By CARL C. FRANCIS, A.B., M.D., Senior Instructor, Laboratory of Anatomy, Western Reserve University, Cleveland, Ohio. London: Henry Kimpton. 1937. Pp. 320. 12*s*. 6*d*.

THOUGH the title of this book would seem to imply biological reference, the work is actually an introduction to descriptive anatomy for the student beginning his study of the body. The author has done his work well with the aid of Prof. Wingate Todd and other well-known authorities. Anatomy is approached mainly from the histological aspect and greater use of embryological detail might have been appreciated by the student anxious to understand the real structure of the parts. There are some excellent illustrations in this volume, and a good glossary and index.

The Subnormal Mind

Second edition. By CYRIL BURT, M.A., D.Sc. Oxon., Professor of Psychology, University of London. London: Humphrey Milford, Oxford University Press. 1937. Pp. 372. 10*s*. 6*d*.

THIS book has been expanded as well as revised in its second edition. Its title should not lead a new reader to expect dogmatic teaching on the recognition of mental abnormality because the distinction between the normal and the abnormal is often artificial or indefinite. There is an illuminating chapter about the normal mind, followed by others on the intellectually subnormal—the defective and the dull or backward—and the temperamentally subnormal as represented by the delinquent and the neurotic. The application of the term "subnormal" to the neurotic is somewhat unusual, but is at least partly justified by the fact that their difficulty is largely one of satisfactory adjustment to environment and so is in some sense a failure in development. When, however, the maladjustment depends more upon circumstances than upon

personality, the term "subnormal" may be open to objection. Prof. Burt has dealt with certain of the types he describes, particularly the backward and the delinquent, in other works, and there is therefore some inevitable repetition.

The book provides a valuable introduction to a study of the mental and nervous abnormalities of both children and adults. Prof. Burt writes as a psychologist but obviously has a sound knowledge of the medical aspects of the conditions treated. He does not, like some psychologists, exclude the factor of heredity in causation. His book is based essentially upon personal experience with cases of all types, particularly among school-children, and he provides in it a basis for the comprehension of conditions which are not even yet adequately dealt with in the medical curriculum.

Synopsis of Genito-Urinary Diseases

Second edition. By AUSTIN I. DODSON, M.D., F.A.C.S., Professor of Genito-Urinary Surgery, Medical College of Virginia. London: Henry Kimpton. 1937. Pp. 294. 12*s*. 6*d*.

THE student's curriculum is now so crowded with special subjects that synopses have become almost essential to him. Genito-urinary surgery, being a specialty that has not long been established, has naturally yielded a recent crop of handbooks in which are compressed all that the student need know. In many of them an attempt has been made to cater also for the general practitioner, but Prof. Dodson has evidently not tried to do this. The fact that a great deal of space has been devoted to pathology and general urological principles, and very little to actual treatment, makes the work more suitable for the student than for the practising doctor. The illustrations, simple line drawings, are adequate if by no means elaborate.

In the new edition have been included such recent advances as the ketogenic diet and the hormonic treatment of retained testis. A discussion of neurogenic and functional disturbances of the bladder is also added to Chapter 12. The book fulfils its function in so far as it is clearly written and well arranged and illustrated.

The 1937 Year Book of Urology

By JOHN H. CUNNINGHAM, M.D., Associate in Genito-Urinary Surgery, Harvard University Post-Graduate School of Medicine. Chicago: Year Book Publishers; London: H. K. Lewis. 1937. Pp. 472. 10*s*.

THE use of year books and annuals is becoming more and more necessary to medical men who wish to keep up to date. As the editor points out in his introduction to this one, some important articles may be buried in the transactions of societies and thus missed even by industrious readers of general and special journals. It is of immense benefit to find noted and summarised within the covers of a single book everything of moment that has been written during the year.

Useful features in this particular year book are the carefully prepared index of subjects and authors and the cross references to matter in previous annuals of the series. These indices give material aid in the preparation of articles or contributions to discussions.

The most notable advances made in urology in 1937 include better methods of dealing with infection and of diagnosing and treating the more obscure cases of renal pain. Our knowledge of the pathology of prostatic enlargement and congenital abnormalities of the ureteric tract has been considerably extended, and we have acquired a more accurate realisation of the indications of transurethral surgery in cases of prostatic obstruction. These advances are set out with precision and judgment by Dr. Cunningham.

Biology for Medical Students

Second edition. By C. C. HENTSCHEL, M.Sc., Senior Lecturer in Zoology, Chelsea Polytechnic; and W. R. I. COOK, B.Sc., Ph.D., Lecturer in Botany, University College, Cardiff. London: Longmans, Green and Co. 1937. Pp. 664. 18*s*.

THE first edition of this book was written mainly for the benefit of students reading for the London First M.B. examination. Since it is anticipated that the London syllabus is shortly to be brought into line with that now adopted by Oxford and Cambridge, a new edition has been compiled which includes descriptions of some extra animal and plant types. The book covers this new syllabus very thoroughly and it would be difficult to discover any omission of facts likely to be required for examination purposes. As stated in the preface, it provides the "necessary information." It would be unfortunate, however, if medical students were to confine their biological reading to this book. It is an admirable guide to practical work but makes little attempt to point out the significance of the facts detailed in relation to general principles or to emphasise the essential unity of the biological sciences, including medicine. This surely should be the main function of a biological course in the medical curriculum, and its fulfilment depends on the proper presentation of the subject.

Medicine

Essentials for Practitioners and Students. Third edition. By G. E. BEAUMONT, D.M., F.R.C.P., Physician to the Middlesex Hospital. London: J. and A. Churchill. 1937. Pp. 780. 21*s*.

A NUMBER of additions to this book in this new edition—the third in its five years of useful life—includes much that is of practical importance as well as some rarities. It is good to find some account of the sulphonamide drugs (though the author does not seem enthusiastic about their merits), of the fuller diets now being given in cases of hæmatemesis, and of the newer insulins. Dr. Beaumont's advice on protamine insulinate differs from that given by some other workers. Dr. Joslin, for instance, who has more than 1000 patients using protamine zinc insulin, finds it adequate for over 80 per cent. of his elderly patients in Boston, but for under 20 per cent. of his young patients; whereas Dr. Beaumont regards the newer insulins as of value chiefly for young people. His opinions are sometimes expressed with diffidence, whereas in fact rather more dogmatism would doubtless be welcomed by the student in certain places. When he is told that such-and-such a treatment "may be tried" his natural question is, "Do you recommend it?" And too often there is no indication in what circumstances the answer would be "Yes." In an age sceptical of therapeutic advances it is satisfactory to note the number (over 20) of new remedies that have had to be added since the last edition of this book, among them mandelic acid, benzedrine, prostigmin, gold salts, and viper venom. The popularity of this book should be maintained by the new edition.

THE LANCET

LONDON: SATURDAY, JANUARY 15, 1938

PREGNANCY TOXÆMIA AND THE PITUITARY

IN the days when the only pituitary preparations available for contracting the uterus were simple extracts of the posterior lobe, the obstetrician employing them sometimes encountered unpleasant or even dangerous circulatory reactions to which he gave the name "pituitrin shock." These reactions were undoubtedly caused by the pressor hormone and little has been heard of them since KAMM succeeded in separating the oxytocic and pressor components of the crude extract. The practising obstetrician will no doubt be glad enough to forget that he was ever haunted by such a spectre, but the more academically minded are still curious to know why some women should react so violently to vasopressin during pregnancy, and the phenomenon has been receiving attention in several quarters.

In Chicago DIECKMANN and MICHEL [1] have compared the effect of subcutaneous pituitrin, in doses of ½–2 minims, on the blood pressure and on water diuresis in normal pregnancy, in pre-eclamptic toxæmia, and in pregnancy complicated by nephritis. They find that the subsequent rise in blood pressure varies in normal pregnancy from 0 to 28 (average 11), in pre-eclampsia from 28 to 80 (average 51), and in nephritic pregnancy from —10 to 18 mm. Hg (average 7·7). The anti-diuretic effect of pituitrin also appears to be greater in the pre-eclamptic than in the other two groups, although here the issue is confused by the probability that any person who is becoming waterlogged would excrete only a fraction of any fluid ingested. The same question has also been studied in Louvain by SCHOCKAERT and LAMBILLON,[2] but they have used intravenous injections and they have included normal non-pregnant women in their survey. The normal woman reacts to an intravenous dose of 3 units of vasopressin by a rise in blood pressure of about 45 mm. Hg, a slowing of the pulse, and a variety of unpleasant subjective symptoms that can be ascribed to the effect of vasopressin on plain muscle in general and on the vessels in particular. In the later stages of normal pregnancy these symptoms are slight or negligible, the pressor response is less (about 25 mm. Hg), and the pulse is accelerated. In pre-eclampsia, in contrast, the blood pressure rises by about 80 mm. Hg, the pulse-rate is greatly reduced, and unpleasant symptoms are noticeable. In nephritic pregnancy, finally, the response to vasopressin is much the same as in normal pregnancy.

Evidently, then, the pre-eclamptic is peculiarly sensitive to vasopressin and it is suggested that this characteristic may be useful in diagnosis. But there has long been a suspicion that over-secretion of vasopressin may be concerned in causing eclampsia, and although any test that promises to distinguish between nephritic and toxæmic pregnancy deserves serious consideration, the risk of precipitating convulsions must be borne in mind. Both the American and the Belgian investigators are evidently alive to the possible dangers of the test and intravenous injection of vasopressin is not likely to appeal to the conservative clinician. Nevertheless it is of no small interest to learn that the eclamptic is hypersensitive to vasopressin. The experiments described elsewhere in this issue by Dr. F. B. BYROM suggest that this sensitisation may be related to the increased secretion of œstrogenic hormone which is characteristic of the period of pregnancy to which eclampsia is confined. BYROM points out, however, that to reconcile eclampsia with œstrin-sensitisation it is necessary to invoke an additional desensitising factor which is present in normal pregnancy and lacking in eclampsia. His work has not yet reached a stage at which it is possible to say whether this presumed missing factor is or is not progesterone, but the clue is a promising one.

HÆMATOMA OF THE RECTUS ABDOMINIS

THE surgeon no longer considers the abdominal wall merely as a barrier which he must circumvent or break through in order to reach the viscera behind it; he recognises it as a region of the body which has its own special problems of anatomy and pathology. A study of the rectus abdominis muscle by CULLEN and BRÖDEL [1] is therefore to be welcomed as an interesting contribution to abdominal surgery. Their paper begins with a detailed but clear review of the anatomy of the rectus with particular reference to its blood-supply. The lower two-thirds of the muscle is supplied by the inferior epigastric artery, which for the most part lies on its posterior surface, not embedded in the muscle, and below the semilunar fold of Douglas is in direct contact with the peritoneum. The whole vascular system is so arranged as to allow the greatest freedom of movement to the muscle, with the least risk of tearing the vessels, and this is particularly necessary in the rectus because of its length.

Hæmatoma is the lesion which has chiefly concerned CULLEN and BRÖDEL, the reason being that it may closely simulate intra-abdominal conditions, especially when it occurs (as it usually does) in the lower part of the muscle where it is next to the peritoneum. The hæmatoma is produced either by a tearing of the muscle itself or of the blood-vessels alone. Direct violence is not the most common cause; as a rule the patient is suffering from some debilitating disease, and a

[1] Dieckmann, W. J., and Michel, H. L., *Amer. J. Obstet. Gynec.* 1937, **33**, 131.
[2] Schockaert, A., and Lambillon, J., *Brux. méd.* 1937, **41**, 1468.

[1] Cullen, T. S., and Brödel, M., *Bull. Johns Hopk. Hosp.* November, 1937, p. 295.

sudden muscular exertion such as coughing ruptures the muscle; sometimes the symptoms come on insidiously. Heart failure and pregnancy are two important general causes, while typhoid fever and influenza may both weaken the muscle locally by producing the hyaline degeneration that is associated with the name of ZENKER. Diagnosis is often difficult, especially when there is no history of trauma or of a sudden onset of the symptoms, and some cause of local peritonitis such as appendicitis or cholecystitis is usually suspected. There is also another class of case in which hæmatoma may be found and in which a special diagnostic difficulty may arise. A local predisposing cause of hæmatoma of the rectus is a previous operation through it or near it, which by setting up adhesions interferes with the mechanism by which the muscle is allowed to move independently of the vessels; in this way a sudden contraction of the muscle may produce a shearing stress which will tear the adherent vessel. A tense, tender, irreducible lump appears in the region of the old scar, and the picture of strangulated incisional hernia is fairly complete.

If the diagnosis of hæmatoma of the rectus can be made with confidence no operative treatment is advisable provided the bleeding has stopped. Usually, however, the diagnosis is established only when the lesion is exposed, and in these cases the clot should be turned out, the bleeding vessels ligatured, and the torn muscle repaired. Rarely, in cases of typhoid and influenza, the hæmatoma becomes infected by the specific organism and the sequel is an abscess.

B.B.S. AND DISEASE

OF late years increasing attention has been paid to the bisulphite-binding substances (B.B.S.) of the blood in relation to vitamin-B_1 deficiency and other conditions encountered in clinical medicine. So far as vitamin B_1 is concerned the experimental foundation for this work was laid in observations in the department of biochemistry at Oxford, the details of which appeared in a lecture by Prof. R. A. PETERS, F.R.S., published in this journal.[1] It suffices now to say that PETERS and H. M. SINCLAIR, and R. H. S. THOMPSON gave proof by in-vitro respiration studies with the brains of pigeons that in vitamin-B_1 deficiency there is a specific accumulation of the intermediary carbohydrate metabolite pyruvic acid ($CH_3CO.COOH$), whereas in the presence of this vitamin pyruvic acid does not accumulate. The implication of this work is that pyruvic acid is a normal stage in oxidation of carbohydrate which accumulates because, in the absence of vitamin B_1, further oxidation cannot take place. Developing this story THOMPSON and R. E. JOHNSON found that in animals suffering from vitamin-B_1 deficiency pyruvic acid collects abnormally in the blood, disappearing upon cure of the animal. It should be mentioned, however, that there is no evidence that pyruvic acid under these conditions is actually toxic. Its presence can be demonstrated as an increase in the value of B.B.S. in the blood, and though the property of binding bisulphite is shared with other ketonic and aldehyde substances, there is a clear proof that in the case of vitamin-B_1 deficiency it is actually pyruvic acid that accumulates. The work mentioned shares its place with other observations upon substances of this type; for instance, PI SUÑER [2] has found pyruvic acid together with methyl glyoxal in certain urines, notably those of diabetics. Further, as PETERS pointed out, A. GEIGER and colleagues have claimed the presence of methyl glyoxal in the urines of infants suffering from vitamin-B_1 deficiency.

Clinical observation in this country led to no finding of increased B.B.S. in the blood except in cases of diabetes where increases might be expected to be due to aceto-acetic acid.[1] But in the Orient, at Shanghai, in excellent work now unfortunately suspended, PLATT and LU [3] obtained evidence in some detail that the results of the animal experiments were applicable to beri-beri in man. They believed, however, that substances other than pyruvic acid, which were estimated by the B.B.S. method, were also increased in the blood, and SHINDO [4] claims the identification of acetaldehyde in the blood of beri-beri patients. Quite recently TAYLOR, WEISS, and WILKINS [5] have reported upon the examination of the bloods of a variety of clinical conditions. Finding that the normal range for B.B.S. is $4{\cdot}7 \pm 1{\cdot}0$ mg. per 100 c.cm. of blood (calculated as pyruvic acid), they consider that all cases showing 6 mg.+ per 100 c.cm. blood can be deemed to have a raised B.B.S. value. According to this criterion, raised B.B.S. were found in 10 out of 19 cases of uncompensated organic heart disease, 18 out of 23 cases of untreated vitamin-B_1 deficiency, 6 out of 9 cases of infectious disease, 7 out of 7 cases of diabetes with acidosis, and 2 out of 4 cases of nephrosclerosis. Treatment of the heart disease led to improvement in the B.B.S., and treatment of the vitamin-B_1 deficiency consistently reduced it. Of substances at present known the most likely to bind bisulphite are acetone and aceto-acetic acid, and in the diabetics there was evidence that the increased B.B.S. value was due largely to these, though pyruvic acid was also demonstrated in the urine in confirmation of PI SUÑER and FARRÁN; it was the investigators' view that the increased B.B.S. was an accurate index of the clinical severity of the diabetes. In heart disease, the raised B.B.S. value was often not due to the ketosis alone. WILSON and GHOSH,[6] in a paper inspired by the work of PLATT and LU, find that B.B.S. is increased in India in epidemic dropsy, anæmia, and splenomegaly as well as diabetes; the greatest increase (up to 8 mg.) was in epidemic dropsy.

It is clear, as TAYLOR and his colleagues say,

[1] Peters, R. A., *Lancet*, 1936, **1**, 1161.

[2] Pi Suñer, A., and Farrán, M., *C.R. Soc. Biol. Paris*, 1936, **122**, 859.

[3] Platt, B. S., and Lu, G. D., *Quart. J. Med.* 1936, n.s. **5**, 355.

[4] Shindo, T., *Hoppe-Seyl. Z.* 1937, **247**, iii.

[5] Taylor, F. H. L., Weiss, S., and Wilkins, R. W., *J. clin. Invest.* November, 1937, p. 833.

[6] Wilson, H. E. C., and Ghosh, B. K., *Indian med. Gaz.* 1937, **72**, 147.

that the increase in B.B.S. cannot be used by itself as a diagnostic test of any one disease. Nevertheless, when combined with attempts to define chemically the substances present, it may throw light upon certain aspects of disordered function. PLATT has already shown its value as an indication of the action of vitamin B_1 in beri-beri.

TUBERCULOUS MENINGITIS

THE pathogenesis of tuberculous meningitis continues to inspire both speculation and research. In 1935 we reviewed [1] it in the light of much work published in the two previous years, especially on the relation of tuberculous meningitis to miliary tuberculosis, and on the possibility of recovery. We drew attention to the difficulty of making a certain diagnosis of meningitis during life, and suggested that the accepted interpretation of changes in the cerebro-spinal fluid might require reconsideration. At that time the old view that tuberculous meningitis is the immediate and typical result of miliary tuberculosis had been severely shaken by the work of RICH and MCCORDOCK, who maintained that meningitis never follows directly from miliary tuberculosis but is always preceded by the formation of caseous tuberculomata in the brain or meninges. Some of the communications that we quoted, especially one by MORRIS SIEGEL, seemed to support this hypothesis, as does the consistent failure of animal experiment to produce meningitis by inoculation of tubercle bacilli into the blood stream. During the past two years further attempts have been made to arrive at the facts, but the results have been by no means uniform. The evidence that A. B. RAGINS obtained from examination of 47 brains did not bear out the opinions of RICH and MCCORDOCK, but pointed to a direct hæmatogenous spread (in 38 of the 47). C. RADMANN, working on a much smaller material, concluded that meningitis occurs both with and without the previous formation of solitary tubercles.

Stimulated by this divergence of opinion on a somewhat fundamental question, Dr. A. R. MACGREGOR and Dr. C. A. GREEN [2] of Edinburgh set themselves the task of answering it by a comprehensive study of 112 brains. They argued that if RICH and MCCORDOCK'S conception holds good, even in a proportion of cases, some victims of tuberculous meningitis must have had an infection of the brain with focal deposits at some previous time. It was therefore desirable to examine brains of sufferers not only from meningitis but from tuberculosis of other organs. When possible the spinal cords were also examined, and the cerebro-spinal fluid was investigated for bacilli. A search for tubercle bacilli was also made in the fluids of 45 patients with suggestive symptoms who did not die of meningitis. The report shows that of the 88 brains examined in which meningitis was present tuberculous infection antecedent to the meningitis was found in 74; and in 4 others there was disease of the spine. Thus in 88 per cent. there was evidence of old foci in or adjacent to the central nervous system. It is of interest that while foci were found in the meninges alone, in the brain alone, and in both together, the first showed the greatest number. Though there remain 10 cases in which careful examination revealed no antecedent focus, the conclusion may be drawn that tuberculous meningitis is most often preceded by a localised infection. The presence of a focus, however, does not prove that it was the source of the meningitis, and MACGREGOR and GREEN accordingly sought confirmation by histological examination of each specimen, accepting as probable sources only those foci that were not encapsuled, were adjacent to the meningeal surface, and were surrounded by recent exudate. By these criteria 10 of the 78 cases were rejected, 58 were regarded as certain, and 6 as very probable. From their histological observations the investigators are convinced that meningitis does not *arise* at the base of the brain, where it is most typically found in well-developed cases. The distribution of the exudate depends in the first instance on the situation of the initial lesion, though later it is concentrated in the basal cisterns.

While the great majority of specimens examined by MACGREGOR and GREEN supported RICH and MCCORDOCK'S view, there were 23 that did not. Further consideration made it clear, however, that neither did they support the hypothesis of hæmatogenous spread; for in 8 of them miliary tuberculosis was absent and in 7 it was very slight. Of the 25 brains that did not show meningitis 11 contained tuberculous lesions: these subjects were mostly children; miliary tubercle was severe in 7 of them and was confined to the nervous system in 3. The cerebro-spinal fluid (usually obtained post mortem by cisternal puncture) was examined in 20 cases of this group, and tubercle bacilli were demonstrated in 10. It is to be noted that bacilli were found in the fluid of 2 subjects whose brains showed no tuberculous lesion. Besides these post-mortem examinations an investigation was made of the cerebro-spinal fluid of 45 patients, 9 suffering from active tuberculosis and the rest reacting to tuberculin. None of them died or developed meningitis; in all of them lumbar puncture was performed because of symptoms of meningeal irritation. In 3 of them tubercle bacilli were recovered by animal inoculation and in 2 others the cytological changes suggested meningitis. MACGREGOR and GREEN'S explanation is that the lumbar punctures and the symptoms occasioning them coincided with the formation of tuberculous foci in the nervous system. While the result of their work leads the Edinburgh workers to support the view of RICH and MCCORDOCK they recognise that this does not account for the close association found between miliary tuberculosis and tuberculous meningitis. The possible ways by which this difficulty may be overcome are shrewdly discussed, and alternative explanations are submitted. Perhaps the most interesting and most novel of these is that the meningitis may at times give rise to the miliary tubercles.

[1] *Lancet*, 1935, **2**, 950.
[2] *J. Path. Bact.* November, 1937, p. 613.

ANNOTATIONS

RAW APPLE DIET AND INFANTILE DIARRHŒA

EVER since Moro and Heisler popularised the old-fashioned South German remedy of raw apples for diarrhœa, workers in different parts of the world have been trying it with varying success. In an Australian discussion[1] Dr. P. A. Earnshaw of Brisbane shows himself an enthusiast for the method, and what makes his results particularly important is the fact that he has had great success with young babies. Older children are apt to respond to several forms of treatment, but anything that would give uniformly satisfactory results in infancy would be very welcome. Earnshaw uses a specially prepared apple powder made from desiccated apples: two level teaspoonfuls are mixed with an equal amount of cold boiled water to make a paste, and the paste is then added to 4 oz. of hot weak tea and stirred for ten minutes. No sweetening material is used for infants under six months; over that age a quarter-grain tablet of saccharine may be added. This quantity of apple-powder mixture is given hourly during the day and every two hours during the night. After twenty-four hours hot boiled whey is used instead of hot weak tea. On the third day the apple-powder paste is added to a hot mixture of a dried lactic-acid buttermilk made with one measure of the milk powder to 4 oz. of water. On the fourth day double the quantity of all ingredients is given every four hours, and from the fifth day onwards the buttermilk mixture is gradually replaced by skimmed milk and then whole milk. At this stage the amount of apple-powder is reduced and gradually it is discontinued.

Other contributors to the discussion were rather mixed in their views. Dr. R. L. T. Grant regards the introduction of apple diet for diarrhœa in infancy as one of the biggest advances he has witnessed, but Dr. N. Cockburn has met with little success in infants under nine months, though the remedy has been very useful at later ages. Most of these Australian speakers seem to be referring to a dysenteric type of disease for which the apple diet is particularly valuable, and it is not quite clear whether young infants with the acute dehydration and intoxication sometimes encountered would respond in the same way. Evidently, however, as Dr. Le Messurier pointed out, this diet reduces the risk of intoxication, since it is almost free from protein.

A SECRET OPERATION

Sir D'Arcy Power, whose articles on bygone surgery have until lately been so regular and attractive a feature of the *British Journal of Surgery*, contributes to an American journal an account of an operation secretly performed on a President of the United States.[2] On June 18th, 1893, Dr. Robert Maitland O'Reilly, medical attendant on Government officials in Washington, was asked to examine the roof of President Grover Cleveland's mouth, and to diagnose the nature of a "rough place" which had probably been present for at least a month. This proved to be an ulcer with some underlying diseased bone, but there was no enlargement of glands. The pathologist reported that the tissue was strongly suggestive of malignancy, the diagnosis was confirmed by Dr. Joseph D. Bryant, and immediate operation was advised. To this the President consented on condition that everything was done in secrecy. At that time the country was passing through a serious financial crisis, and the gold reserve in the Treasury was sinking dangerously. Cleveland had been kept desperately busy, so that his general health, and particularly his teeth, had been neglected.

The operation took place on July 1st on the yacht *Oneida*, which was lying at anchor a considerable distance from the New York battery. A day beforehand Dr. Edward Gamaliel Janeway, one of the most eminent diagnosticians of the time, went on board to examine the chest of the distinguished patient, who had arrived from Washington. The anæsthetic caused some anxiety, for the President was 56 years old and very fat. His neck was especially short and thick. While the yacht was going up the East River at half-speed Dr. Ferdinand Hasbrouck, a dentist, gave nitrous oxide and extracted the upper left bicuspid teeth. Dr. O'Reilly administered ether, while Dr. Bryant incised the roof of the mouth, with Dr. William Williams Keen and Dr. John F. Erdmann acting as assistants. The entire left upper jaw was removed from the first bicuspid to just beyond the last molar and close to the midline, but the floor of the orbit was left intact. The antrum was found to be filled with a gelatinous material, afterwards reported to be sarcomatous. The patient made a good recovery. The absence of an external scar—the entire operation had been done within the mouth—and the preservation of the normal voice and of the normal position of the eye, together with a skilled prosthetic appliance, helped to keep the operation a secret. Cleveland died fifteen years later without any recurrence of the growth.

THE IMMUNISING ANTIGENS OF SHIGA'S BACILLUS

THE Gram-negative bacilli of the typhoid-dysentery group were among the first organisms to be studied thoroughly by the agglutination reaction, and in consequence a large number of bacilli in the group have been described in terms of separate antigenic components. But owing, perhaps, to the relatively clear-cut answers given by agglutinin-absorption studies on these organisms, the analysis of the bacterial bodies into soluble antigenic components has not yet reached a stage comparable, for example, with that of some of the pneumococci and streptococci, and of the tubercle bacillus—all organisms whose agglutination reactions yield but little information about their antigenic structure.

The several attempts made to isolate antigens from the Gram-negative bacilli made it evident that, as in the pneumococcus, the specificity of their immune reactions depended in part upon complex polysaccharide substances. Dr. W. T. J. Morgan has previously reported[1] the isolation of a specific polysaccharide from Shiga's dysentery bacillus, which, though reacting with homologous antibody both in vivo and in vitro, was not itself fully antigenic after it had undergone the treatment involved in its isolation. Since then, starting from the observation that bacterial and other polysaccharides were fairly soluble in various glycols, but that proteins were almost insoluble, he has developed a method for extracting bacteria that may prove applicable to

[1] *Med. J. Austral.* 1937, **2**, 756.
[2] Power, D'A., *Surgery*, 1937, **2**, 780.

[1] Morgan, W. T. J., *Brit. J. exp. Path.* 1931, **12**, 62; *Biochem. J.* 1936, **30**, 909.

a number of species.[2] By extracting acetone-dried Shiga bacilli with ice-cold water-free diethylene glycol, he obtained an apparently non-protein substance capable of inducing in rabbits a copious formation of specific antibodies. The sera of rabbits immunised with the substance also agglutinate sheep red cells—i.e., heterophile antibodies are produced. On acid hydrolysis, the antigenic material yields a lipoid-containing substance, and the specific polysaccharide previously identified. The power to stimulate heterophile antibody is destroyed by the action of mild alkali, a treatment that has little effect on the polysaccharide part of the antigen, and it appears that the heterophile antigenic structures are present in the as yet unidentified component of the Shiga antigen.

The immunising antigen of Shiga's bacillus is similar in its general properties to the antigens isolated from Gram-negative bacilli by Boivin and his co-workers, and from *Bact. aertrycke* and the typhoid bacillus by Topley, Raistrick, and others.[3] The successful isolation of these antigens seems dependent on the avoidance of any gross chemical treatment of the bacilli. The typhoid antigen was resistant to the action of trypsin, and was isolated from a tryptic digest of dried bacteria that had undergone no other treatment. The introduction of diethylene glycol as a selective antigenic solvent marks another advance in the technique of immuno-chemical analysis of bacteria.

THE INCREASE IN LONGEVITY

LAST week (p. 106) an American correspondent referred to some interesting observations made on the expectation of life by Harold F. Dorn, statistician in the United States public health service. Dorn calculated that a child born in the United States in 1933 could expect to live 12 years longer than his parents could have expected at their birth, but that to achieve the same increase during the next generation would require every new-born infant to live until nearly age 55, unless there is a sharp decrease in mortality at older ages. The prospect of either event obviously does not appear at all likely. England and Wales stands in much the same position, and it is perhaps not generally realised how much of the increase in the expectation of life in the past 40 years is due to the reduction of mortality in infancy and early childhood. The "expectation" is, of course, only a technical (and rather misleading) term for the *average* length of life that would be lived by a cohort of babies if they were exposed throughout life to the death-rates at different ages prevailing in a given place and time. For instance at the death-rates of England and Wales in 1930–32 the average length of life of males would be 58·74 years, of females 62·88 years. At the death-rates of 1910–12 the corresponding averages are 51·50 and 55·35. In other words, the expectation of life at birth has increased in an interval of twenty years by rather more than 7 years for each sex. On the other hand, if we survey the survivors at age 10 and measure their expectation, or average length, of life—i.e., subsequent to that age—then the increase between 1910–12 and 1930–32 is rather less than three years. At age 60 the increase is only two-thirds of a year for males, a year for females. At age 80 the expectation is actually a fraction lower in 1930–32 than in 1910–12. In other words, we have more persons kept alive until late adult life, but they can then expect to live little, if any, longer than their predecessors. Further reductions in the infant mortality-rate will, of course, somewhat extend the expectation of life but not, in accordance with Dorn's calculations, a very great deal. Can we hope to reduce the death-rates at older ages—from cancer, diabetes, heart disease, nephritis, and cerebral hæmorrhage? It seems more likely that we shall observe a slackening rate of improvement in the expectation of life in the next generation.

[2] Morgan, W. T. J., *Biochem. J.* 1937, **31**, 2003.
[3] Topley, W. W. C., Raistrick, H., Wilson, J., Stacey, M., Challinor, S. W., and Clark, R. O. J., *Lancet*, 1937, **1**, 252, 274.

CONCERNING MUMPS

BY "mumps," without qualification, is understood the affection of the parotid glands described by Hippocrates; but Dr. R. E. Smith[1] does well to remind us that any or all of the salivary glands may be involved and he has himself described a small epidemic in which the submaxillary glands were those chiefly affected. There can be little doubt that unrecognised solitary submaxillary or sublingual mumps may be the starting-point of an outbreak of parotitis. The usual complications of mumps may be secondary to inflammation of any of the salivary glands and it is possible therefore that reported examples of primary orchitis have really been secondary to unrecognised atypical attacks of this disease. From an analysis of the records, Smith concludes that the usual incubation period is 18 days (which agrees with that arrived at by Orr,[2] who applied the method of the periodogram); incubation periods longer than 25 days or shorter than 14 are rare. Accepting the results of an investigation carried out by the Clinical Society of London (1893), Smith says that mumps is infectious not more than 24 hours before the appearance of any swelling; but this is not the universal opinion. Rolleston[3] prefers to say "probably at least 24 hours," and although the evidence is not strong it has been asserted that mumps may be infectious towards the end of incubation. It is certain that in some cases prodromata such as sore-throat, coryza, stiffness of the neck, malaise, and pyrexia may precede the swellings by as long as 3 days, and such cases must be regarded as potentially infective although, as Smith remarks, mumps is probably the least infective of the common specific fevers of children. The duration of infectivity is doubtful; in Smith's experience at Rugby boys discharged a week after the subsidence of the last swelling or a fortnight from the onset (whichever is the longer) have given rise to no further cases, and he thinks it probable that these times could be shortened. Rolleston, indeed, says that swelling and infectivity disappear simultaneously. It may be added that the periods of segregation of other of the common fevers might be abbreviated with advantage not only to the patient but to the public purse. Rubella is a case in point; isolation for 5 days is ample. Since contacts rarely develop mumps, and originators of epidemics rarely give a history of contact, Smith believes that quarantine for contacts is wasteful. Provided that diagnosis and segregation are prompt, this will be generally agreed; mumps is not readily conveyed from person to person by droplet spray. It must be remembered that in the early stages the infective agent, generally accepted to be a filtrable virus, may be transmitted upon utensils or the hands of attendants from one child to another

[1] Smith, R. E., *Guy's Hosp. Rep.* 1937, **87**, 447.
[2] Orr, H., *J. R. Army med. Cps*, 1918, **31**, 480.
[3] Rolleston, J. D., Acute Infectious Diseases, 2nd ed., 1929, p. 234.

in a ward; nevertheless to quarantine the ward is rarely worth while.

Smith also discusses prophylaxis by means of convalescent serum and draws some analogies from the seroprophylaxis of measles. He concludes that given in small doses at the beginning of the incubation period mumps serum "appears to have definite preventive powers." But here again the relatively low infectivity of mumps must be borne in mind, and accounts of the successful prevention of mumps should for this reason be accepted with reserve. It is a question not of contact merely but of *effective* contact, and the same caution must be observed in assessing the results of the seroprophylaxis of measles under different degrees of propinquity. The intimate repeated familial contact that obtains in the tenement is by no means the same as the exposure—contact is hardly the word—that is likely in a children's ward. Allowing the potential prophylactic value of convalescent mumps serum, Smith makes the important observation that by artificially postponing an attack of mumps until the age of puberty there is a danger of encouraging serious complications, such as orchitis, rare or unknown among young children. Provided the correct dosage can be estimated, the use of serum to ensure a mild attack, from which the patient obtains lasting immunity, will usually be preferable.

THE CARE OF MENTAL DISORDER IN LONDON

THE immense scale of the coördinated provision made by the London County Council for the recognition and treatment of every sort of mental ill health is manifest in the annual report just issued [1] of their mental health services. The 33,000 beds in various mental hospitals represent only a part of what is done. For the defective there is a system into which enter school medical officers, supervisors who visit the patients' homes, occupational and training centres, and a venereal disease unit; attempts are made to place juvenile defectives in remunerative employment, and advice is given to the court about supposed defectives who have been charged with criminal offences. The work for mental disorder, including neuroses and the behaviour problems of children, interlocks with that of the general health department in such matters as the provision of psychiatric out-patient clinics at some of the general hospitals and in the psychiatric staffing of observation units. The trends towards coördination and grading are indicated in the decision that St. Ebba's Hospital (formerly called Ewell Mental Hospital) is to serve as a second string to the Maudsley Hospital by receiving, as far as possible, patients with a hopeful prognosis, but for whom a longer term of treatment is likely to be needed than for those generally regarded as suitable for the Maudsley Hospital, where accommodation is limited. At St. Ebba's Hospital an interesting experiment is being tried: patients who have recovered are taught a more congenial or suitable trade than they had formerly, in order that they may return to ordinary life with less risk of social difficulties and a further breakdown. The decision to omit the word "mental" from the titles of any hospitals in the service is welcome. Another desirable alteration in nomenclature would be the abandonment, in the report, of the old-fashioned diagnostic classification that has for so long been officially required by the Board of Control.

The statistics published in the report show some curious features. For example there is an extraordinary fluctuation in the number of new cases annually notified for the first time under the Mental Deficiency Act, 1913, whereas each year the number of defectives who require institutional care increases by about 250, and will probably continue to do so. In the greater part of South London an average of 6 patients is directly admitted to mental hospitals for every 10,000 of the population, yet in a part of North London the average is as high as 10; in some districts south of the Thames nearly a third of the patients admitted come into the mental hospital voluntarily, whereas in other districts of London only a sixth or an eighth do so. Between Jan. 1st, 1936, and Jan. 1st, 1937, "the unsatisfied demand for beds was mainly for cases of senile dementia." It must be a matter of conjecture to what this shortage is due: partly perhaps to the increasing number of elderly persons in our population, partly to changes in housing and social conditions which make people reluctant or unable to look after their failing relatives at home. Perhaps it is mainly an administrative question of making provision for the better nursing and care of senile patients formerly housed in general hospitals and institutions for chronic illness. On almost every page of this report the bald numbers raise questions which bald numbers cannot answer. Interesting and informative as are the detailed figures published, they are really only raw material on which the psychiatrist, the statistician, and the sociologist must get to work; for such purposes the London County Council can supply unsurpassed factual material, to be compared with that of Massachusetts or New York State.

The activities in psychiatric teaching and research are chiefly carried out at the Maudsley Hospital, a school of the University of London, where the creation of university chairs in psychiatry and in the pathology of mental disease (filled respectively by Prof. Edward Mapother, the medical superintendent, and by Prof. F. L. Golla, the director of the central pathological laboratory) establishes the coöperation needed for post-graduate education. Other signs of this development are the appointment of Dr. Aubrey Lewis as clinical director at the Maudsley to take charge of the clinical training of post-graduates and the coördination of research; the additional facilities provided at certain observation units for the fuller training of physicians and social workers, and the biennial refresher course for psychiatrists. Prominent among the research activities mentioned in an appendix is the work done by Mr. Grey Walter, partly in the central laboratory at the Maudsley and partly at Maida Vale Hospital for Nervous Diseases, on the application of the electro-encephalogram to the localising of cerebral tumours and the treatment of epilepsy. Investigation into prophylaxis of G.P.I. was being, during the year under review, conducted at the centre at Horton Hospital, and by Dr. A. Beck, one of the research workers at the central laboratory; special studies on mental defect and its pathological basis, on the effects of benzedrine, and on the inheritance of mental disorder were also carried out.

[1] Annual Report of the London County Council, 1936. Vol. VI. London: P. S. King and Son. Pp. 69. 1s.

THE VALUE OF TOTAL OPIUM ALKALOIDS

SINCE it was first recommended by Sahli thirty years ago, a mixture of the hydrochlorides of the opium alkaloids in their natural proportions has been used extensively, under the name of Omnopon or Pantopon, in place of morphine. This preparation has been said to cause less nausea and vomiting and respiratory depression than morphine, and to be

more effective in relieving pain and relaxing spasm of plain muscle. Hayman and Fox [1] of Cleveland have now compared its efficacy with that of morphine sulphate in pain after operations, in renal colic, pleurisy, peritonitis, facial herpes, and other conditions, and in restlessness due to cardiac failure. Hypodermic tablets were prepared which were indistinguishable as regards their shape and colour, so that neither the patient nor the nurse knew which was being given. The effects of about eighty injections of omnopon were compared with those of about eighty injections of morphine sulphate. There was great variation in relief that the same patient got at different times from the same drug, but complete relief within half an hour was obtained from roughly 60 per cent. of injections with morphine sulphate gr. $\frac{1}{4}$, from 60 per cent. with omnopon gr. $\frac{1}{3}$, and from 45 per cent. with morphine sulphate gr. $\frac{1}{6}$. Nausea was noted in one patient after morphine and in three after omnopon, but there was no obvious difference in the amount of respiratory depression. Omnopon contains 50 per cent. of anhydrous morphine hydrochloride, and owing to the water of crystallisation in morphine sulphate there is only about gr. 1/25 less morphine alkaloid in gr. $\frac{1}{3}$ of omnopon than in gr. $\frac{1}{4}$ of morphine sulphate. It seems then that the effects of omnopon —at any rate under the conditions of this test— do not differ appreciably from those of the morphine it contains.

INVESTIGATION OF ASTHMA

THE latest report from the Asthma Research Council [2] suggests that the attack on the allergic group of diseases is proceeding by small advances on a wide front rather than by any spectacular turning movement. Workers at Guy's Hospital have continued to review the efficiency of their armament by comparing the results of treatment with specific agents, such as sensitising proteins and autogenous vaccines, and those obtainable with indifferent agents such as sterile saline solutions. An allergen-free chamber has been installed and should be of value in simplifying the study of individual patients. Both at Guy's and at St. Mary's Hospital further investigation has been made into the psychogenic factors in asthma, and though there may be difference of opinion whether these factors are primary or secondary there is agreement as to their great importance. At St. Mary's much interesting work has been done on hay-fever, which is in many respects the disease best suited for research on allergy. It has been shown that hay-fever patients may be sensitive to, and may be relieved by treatment with, exotic pollens with which they have not come in contact, such as that of sugar-cane. These tests are taken as additional proof of the homogeneity of the pollen antigen and the uselessness of testing patients with a long series of grass-pollens to see "which is causing the mischief." More has been learnt about the chemical nature of the toxic substance in pollen and about the properties that develop in the serum after protective inoculation. Bacterial allergy and the toxins associated with focal sepsis have also been studied, but though a toxin indistinguishable from that present in the exudates and blood-serum in eczema, and in the blood-sera in other allergic conditions, has been detected in very many inflammatory conditions and in connexion with various septic foci, no broad ruling on the relation of "allergy" to "immunity" is at present possible.

At King's College Hospital remedial exercises have been under study since 1932 and the results are now being assessed for publication. All patients who have suffered from asthma for any length of time tend to develop a wrong way of breathing which increases the functional disability of the lungs. When they can be taught to acquire a perfect breathing technique about 85 per cent. of them show improvement. A revised edition of "Physical Exercises in Asthma" has just been issued at a price of one shilling, and any profits from this go to the Council's research fund. At Birmingham General Hospital and at Guy's Hospital workers have studied the value of the erythrocyte sedimentation reaction in prognosis and differentiation of cases of asthma. Work of a more fundamental experimental nature has been carried out at King's College and at St. Bartholomew's Hospital. At King's College the spontaneous variability of hypersensitivity has been studied and a number of chemical substances have been tested for their ability to reduce hypersensitivity in the isolated lungs of animals. At St. Bartholomew's Mr. J. L. D'Silva, Ph.D., has continued his important experiments on the mobilisation of potassium from the liver into the circulation after intravenous injection of potassium.

A number of promising openings have thus been made and it is a pity that others have been checked by shortage of funds. The treasurer hopes that all asthma sufferers who read this report will support the research work by sending donations; and when one compares the small income and disinterested service of the Asthma Research Council with the large sums spent on proprietary remedies for the disease, one trusts that this hope will be fulfilled.

ARE PROSECUTIONS FOR POISONING FAIR?

THE challenging paper on prejudicial assumptions in poison cases, read by Miss Alice Raven before the Medico-Legal Society last June, has now been republished separately.[1] Miss Raven finds the atmosphere of a criminal trial on a poisoning charge rather unfair. A case of poison is not necessarily a case of poisoning. Counsel need not keep talking about what would be a "fatal dose." She criticises certain traditional or customary assumptions to which both the prosecution and the judge give too ready a currency and which the defence fails to dissipate. Miss Raven takes, for the sake of argument, arsenical poisoning. Five points, she says, are likely to be too readily assumed. First, if post-mortem examination shows a sufficient amount of arsenic in the body to cause death, it is assumed that death was due to arsenical poisoning. Secondly, if there have been previous illnesses with similar symptoms, these previous illnesses are assumed to have been due to arsenical poisoning. Thirdly, if arsenic is found in a body it is assumed to have been administered with intent to murder. Fourthly, it is assumed that the person who has the best opportunity is the person who administers the arsenic; in other words, opportunity is deemed evidence of guilt. Lastly, a supposed benefit accruing to the person who has the best opportunity is assumed to be a sufficient motive for ending the life of the deceased. These five assumptions, Miss Raven observes, ignore the numerous accidental ways of taking arsenic. She reminds us of the known instances of arsenious oxide being used in error in preparing baking-powder, sugar,

[1] Hayman, J. M., jr., and Fox, H., *J. Amer. med. Ass.* Nov. 27th, 1937, p. 1814.

[2] Report of Progress for the year ended Oct. 31st, 1937. Copies can be obtained from the Asthma Research Council, c/o King's College, Strand, London, W.C.2.

[1] Westhope and Co., London, 6*d*.

salt, cream of tartar, flour, and meal. There may, she says, be inhalation or contact from arsenical wood preservatives, fruit-tree sprays, or sheep-dip; chronic arsenical poisoning can come from the fine dust of wallpapers coloured with the green of aceto-arsenite of copper. There may be arsenic in the soil, arsenic in the glucose from which beer is brewed, and natural arsenic in plaice or soles, oysters or prawns. Miss Raven analyses two well-known poisoning trials in the light of her thesis, the Liverpool case of Mrs. Maybrick in 1889 and the Dorset case of Mrs. Bryant in 1936. The conviction of Mrs. Bryant depended in part upon an empty tin of weed-killer which the police recovered, rusty and battered, from a rubbish-heap. Analysis of scrapings from the outside and inside of this tin disclosed 5·8 per cent. of arsenious oxide. Sifted ashes from the grate of Mrs. Bryant's wash-house copper were found to contain 149 parts per million of arsenic, and the experts said that the normal arsenic content of ordinary coal ash would be about 100 parts per million less. The Court of Criminal Appeal rejected an application by the defence to call further expert evidence which might have put the normal arsenical content of coal ash higher. Bryant, says Miss Raven, worked on soil which contained arsenic. Daily and hourly he would be breathing it in and absorbing it through his skin in minute quantities of dust. This might have explained the arsenic in his body, which might also have been due to arsenical wallpaper in the cottage or to his having handled sheep-dip, or to his being (as she suggests that many farm labourers are) an arsenic-eater. In the result she concludes that it is easily possible for a person accused of poisoning to be hanged on insufficient evidence, and she demands a more rigorous standard of proof of guilt in such cases in future.

When anyone is charged with a horrible offence, prejudice is easy. The mere recital of the charge will make a juror instinctively think what a brute the accused must be. The crime of poisoning is, according to a statute of Henry VIII, of all kinds of murders specially to be detested and abhorred. It was described in the Act of 1351 as "in this Realm hitherto, our Lord be thanked, most rare and seldom committed and practised," and, when the Bishop of Rochester's cook "of his most wicked and damnable disposition did cast a certain venom or poison into a vessel replenished with yeast or barm" so that many died of poisoning, the crime of murdering by poison was exalted into treason and the punishment was ordained to be that of boiling to death. The Bishop's cook was duly boiled to death at Smithfield and a poor woman at King's Lynn, accused of having poisoned her husband, was soon afterwards punished in like manner. To-day, with better scientific knowledge, a less ferocious law, and a system of procedure which gives ampler opportunities to the defence, we may hope that there is less prejudice and fairer trial. But Miss Raven's warning deserves attention though possibly she under-estimates the insistence with which her points have been taken by defending counsel. The suggestion that the viscera of an exhumed body have been impregnated with arsenic from the surrounding earth has certainly been vigorously pressed in more than one recent trial.

THE NEW ST. GEORGE'S

IN their plans for rebuilding St. George's Hospital the authorities have kept two aims in view—to enhance its function as a teaching hospital and secondly to combine economy of space with room for expansion if necessary. The same generous proportion of 330 beds for 100 students is to be maintained, for there are to be 520 beds in the hospital, with 180 members of the medical school. With the same object and with possible changes of emphasis in the medical curriculum in mind, the special departments will be large enough for teaching purposes, and the increase in the number of beds is chiefly accounted for by the new wards for psychiatry (48 beds), obstetrics (48 beds), orthopædics and fractures (36 beds), and dietetics (24 beds). The total includes 51 private beds and cots and also 68 contributory beds—mainly in 2-bed units related to the main wards and thus available for teaching. These are intended for patients who can pay the whole cost either themselves or through contributory schemes. The general medical wards have 90 beds, including 8 for skin disease, and the surgical 120; there are also 16 beds for isolation and venereal cases. The main wards, each in charge of a sister, contain 30 beds, in two equal units for male and female patients. A unit consists of a large 12-bed ward which is subdivided by low glazed screens into bays of 4 beds arranged parallel to the windows. This will combine the maximum of air, light, and privacy with the minimum waste of the nurses' energy. There is also in the unit a smaller ward for 2 contributory patients, the remaining bed of the 15 being in a separate isolation ward. The two units have a common kitchen, general utility and clinical rooms, but different sanitary offices. A clinical theatre and the four main operating theatres will be placed together in a group. The ophthalmological, ear, nose, and throat, and gynæcological wards will have appropriate examination rooms and minor operation theatres. The fracture clinic will be part of orthopædic wards, and the cardiographic department, the chest clinic, and the dietetic department (with metabolic clinic and diet instruction room) are to be in close association with the medical wards. A new departure will be a department for research in industrial medicine. The plan used for the new Westminster Hospital of placing out-patient clinics in relation to the corresponding wards on each floor is not being followed, on the grounds that it will be uneconomical in staffing and in space; it is considered that a single well-planned room can be kept in constant use by many different clinics. The hospital building is to occupy a large site in the same position at Hyde Park Corner, and to make space a number of houses now used for nurses' quarters will be pulled down. It is to be 40 ft. higher and it will be faced with Portland stone to harmonise with other notable buildings in the neighbourhood. The competition for the selection of architects is now open until the end of August. Messrs. Lanchester and Lodge have been appointed assessors.

COURSE ON MENTAL DEFICIENCY.—Lectures and clinical instruction for medical practitioners on mental deficiency and allied conditions have been arranged by the Extension and Tutorial Classes Council of the University of London in coöperation with the Central Association for Mental Welfare. The course is intended for qualified medical practitioners who are concerned with the care of subnormal or abnormal persons. The first part will be held from March 28th to April 9th, when Dr. A. F. Tredgold will lecture on mental deficiency, Dr. A. Carleton Williams on administrative procedure in the ascertainment and treatment of the mentally defective, Dr. Henry Herd on the work of the school medical officer in relation to the mentally defective, and Miss Lucy Fildes, Ph.D., on the psychology of mental defectives. The second part will be given in the autumn. Further information may be had from Miss Evelyn Fox, University Extension Department, University of London, W.C.1.

SPECIAL ARTICLES

CLINICAL EXAMINATION AND DIAGNOSIS IN CARDIOVASCULAR DISEASE

By Terence East, M.A., D.M., F.R.C.P.

PHYSICIAN TO KING'S COLLEGE HOSPITAL, LONDON, AND THE WOOLWICH MEMORIAL HOSPITAL

The method of clinical examination of the heart which is taught to students is no doubt founded on long-established practice and tradition. One cannot but feel, however, that many candidates who present themselves at clinical examinations have not derived as much benefit from studying these methods as might be hoped. When they go into practice, later on, one still meets with examples of diagnosis, or misdiagnosis, which show that the difficulties of clinical examination of the heart have never been successfully overcome. Anything that would simplify or systematise the elucidation and interpretation of physical signs might be useful.

WHAT THE STUDENT MUST LEARN

The expert, of course, can develop his own routine methods, choose his short-cuts, and learn his own tips. Lack of skill in the physical examination of cardiac cases really dates from early difficulties never being surmounted. Six months' clinical clerking is not long for a student to train his senses in the technique of inspection, palpation, percussion, and auscultation.

No doubt the two chief difficulties which confront the student are, first, the gaining of skill in these arts, and secondly, the mental synthesis of the data, which should have been correctly obtained, into a diagnosis.

And there are so many data in a cardiac case. As others have urged (Sir Thomas Lewis, and the American Heart Association), a cardiac diagnosis must cover at least several points, if it is to be at all comprehensive or practical. I will return later to this.

Perhaps one of the difficulties lies in this advantage that we have—we can obtain more accurate data about the function of the heart than of almost any other organ; and it is a complicated double pump at that. I refer to clinical means alone, and when we call to our help radioscopy and electrocardiography, and the study of the circulation-rate, our information is certainly abundant.

The ritual of inspection, palpation, percussion, auscultation is founded no doubt on a good plan. It encourages at first a general survey, and then focuses down on more local points. But the various data the eye obtains have little to do with one another: the state of the veins in the neck, the site and extent of the apex-beat, if visible; and any abnormal movements, distended vessels and so on, that may be seen. *Palpation* examines the movements, may detect thrills, feels the closure of valves.

The value of *percussion* of the heart is a vexed subject in some quarters. I will not discuss its failings and limitations, but I do think that a student should be trained to use his fingers in this way—he can abandon it later if he finds it of no help. He may at first find it useful to confirm his findings on palpation. Of course he must learn its limitations, and where it may fail or be deceptive. While on this topic one might suggest that it was time the curious little diagram $\frac{\text{iii}}{1\,|\,3\frac{1}{2}}$ was abandoned. I am afraid that it conveys little, and may introduce erroneous ideas about the size and shape of the heart. Not that I think it a waste of time for the student to try to diagnose cardiac enlargement. He must train his senses for this, and realise when they will fail him and when he can rely on them. The correlation of skiagrams and clinical examination will help him here in his training. *Auscultation* is mainly concerned with the study of the heart sounds and any murmurs, which may or may not indicate valvular disease or other pathological defects. Perhaps auscultation is the most difficult art the student must learn. Auditory data are always most difficult to interpret. The hearing seems to vary more in keenness than the sight or the touch. Sounds are remote and impersonal. Auscultation provides many data for mental synthesis. One wonders whether the ritual of auscultation, first at the apex, then at the pulmonary area, and then at the aortic area, and then at the tricuspid, is the best means whereby the student may collect them.

AN ANATOMICAL OR A PHYSIOLOGICAL BASIS?

It seems, in fact, that the data to be obtained by the senses have to be fitted into a scheme of examination which depend only on the methods employed; would it not be better to devise some scheme of examination to fit the data to be obtained? It might be easier for the student to have some scheme in his mind which would help him to find what he is seeking. At present he seeks it at random, and then has to try to put together what his senses have told him. Finally, when one comes to think of it, our present method of clinical examination of the heart lays very little stress on the detection of heart failure. This is really the most important thing of all: it is for this that his patients will come to him later on. It is true that the first indications of cardiac inefficiency lie in the patient's symptoms. One cannot speak too strongly of the necessity of a full and careful investigation of dyspnœa, indicating inefficiency or failure of the extracardiac circulation, or of precordial pain showing inefficiency of the intracardiac circulation.

Unfortunately the examination bogy casts its shadow before. A large proportion of the cases at a clinical examination are patients with "well-compensated" valvular disease. The student is often content if he can diagnose them and no more. Detection of failure of the circulation is in consequence overlooked. An *anatomical* basis of examination of a cardiac case is hardly satisfactory. But where we have an organ whose function can be so readily studied and so intimately examined, a *physiological* basis seems to offer a scheme into which the numerous data of clinical examination can be fitted. It has the advantage of linking up, at the outset, a student's physiological work with his clinical.

WHAT THE STUDENT SHOULD DO

The scheme I describe is one which I have used for several years, particularly in demonstrating to students the phenomena of circulatory failure. In such cases it seems to be of the greatest use, but it can of course be applied to all patients with heart disease.

The idea is to follow the circulation of the blood—ride on a red corpuscle as it were—from the periphery to the heart, through the lungs, to the heart again, and then out into the arterial system. If one does this one impresses on the student that the thing that matters, first and foremost, is how the circulation of the blood is being maintained by the heart. He will then include in his study any lesion in the heart that may affect the circulation. Let him start at the periphery, noting cyanosis in the skin of the hands, ears, face, feet; œdema; the state of the capillaries and veins; and then let him note the veins in the neck, their fullness, taking into consideration the patient's degree of recumbency, and their degree of pulsation. He may now palpate the liver and feel for the spleen, looking for ascites and watching the effect of abdominal pressure on the filling of the external jugulars (if this is considered to be a useful practice).

Now he passes into the right auricle. We tell him it lies too deep for its enlargement to cause dullness as a rule. He should however look for dullness to the right of the sternum, which may be caused by it or other things. Next he should auscultate the tricuspid valve, and realise that it rarely produces any distinguishable murmurs. Then he should palpate over the right ventricle, and also note any bulging—it is surprising how few students know these cardiac surface markings—and then over the conus arteriosus, using finger-tips and palm. Then auscultate the pulmonary valves. Here, of course, the blood eludes us. But he must examine the lungs for œdema and the pleural sacs for hydrothorax. Estimation of the circulation-rate is a simple and practical way of demonstrating slowing of the pulmonary circulation. It may well find a part in any clinical examination, or at least in the demonstration of circulatory failure, due discretion being used. The left auricle is beyond our senses except in very rare cases of gross enlargement, unless we use X rays. He can then auscultate at the apex to observe the function of the mitral valve, and listen to the mitral first sound. The left ventricle now concerns him: the site, force, extent, rate, and rhythm; palpation will possibly suffice for most of these—percussion of the left border is held by some to be superfluous. In particular, in studying the mitral first sound, gallop rhythm should never be overlooked. Now he may pass up through the aortic valves, using auscultation, and palpation for thrills, and finally into the aorta. Here again he must glean what scanty data his senses may give him as to its enlargement. Finally he passes out into the arterial circulation: he palpates such arteries as he can get at (not omitting those of the feet), examines the only arterioles he can see, with the ophthalmoscope, feels the pulse, and takes the blood pressure, and may detect alternation. There is a good deal to be said for estimating the blood pressure at the end of clinical examination.

By this method the importance of the circulation is borne home to the student. He can always go back over any point that is obscure, or to correlate one finding with another. But he has a physiological scheme to work on, and on which to hang his data.

WHAT THE DIAGNOSIS SHOULD COVER

Finally, I would emphasise once more, as others have done, the wide scope of diagnosis in heart disease. The diagnosis, or better still diagnostic summary, should always take note of its data under these four headings. It is not of much importance in what order they come, provided they are all included.

1. Efficiency of heart and circulation.
2. Pathological changes (myocardial, valvular, pericardial, arterial).
3. Rhythm.
4. Ætiology.

If these four points are kept in mind, little will be overlooked, and the student will have grasped the important things about the case. Nothing is more interesting and valuable than the application of instrumental methods in the diagnosis of cardiovascular disease. But we must use them to help the student to sharpen his senses and to be able to rely upon his unaided perceptions. It is pitiable when a man says he cannot diagnose some arrhythmia because an electrocardiogram is not available, or gauge the size of a heart unless a skiagram is taken. He must know when the well-trained senses can help him no further. Because they cannot tell him everything, there is no reason why he should not master the difficult art of training them well.

UNITED STATES OF AMERICA

(FROM OUR OWN CORRESPONDENT)

NEONATAL MORTALITY

STUDIES in neonatal mortality have hitherto been made on a small scale, the data being collected painstakingly for the purpose. In a paper presented to a joint meeting of the American Statistical Association and the American Sociological Society on Dec. 28th at Atlantic City, Jacob Yerushalmy, Ph.D., made use of a novel and successful attack. Dr. Yerushalmy is on the staff of the New York State department of health and has had access to the birth and death records of this large State (outside of New York City). Excluding 16 deaths of unidentified infants and 1 neonatal death for which no record of birth could be found or secured, and excluding also births of non-resident mothers, Dr. Yerushalmy had records of 82,138 births and 2566 neonatal deaths in this population for the year 1936. Data from birth and death certificates were then assembled on a single set of punch-cards, by manipulation of which a most interesting series of tables has been constructed.

A number of observations made previously, though upon much smaller series, have been confirmed. Neonatal mortality was found higher among males than among females for every order of birth. First-born infants had a considerably higher neonatal death-rate than second-born infants (32·2 as compared with 26·1). Thereafter the death-rate rose with the order of birth until for tenth and later births it is found to be 54·2.

These facts will surprise no one. We are prepared also to learn that premature babies survive less often than full-term infants. However, the very great difference in risk as shown in the following Table certainly deserves attention. These figures fully justify the campaign which Dr. Julius Hess is waging in this country for special facilities for the care of the prematurely born.

So far Dr. Yerushalmy's statistics confirm existing faith. But his method of attack has also uncovered two correlations that appear to have a profound biological significance. He finds that in his material there is an association between the power of survival of the offspring with the age of each parent at the time of the infant's birth. This relationship is not simply due to the fact that older mothers are more liable to have borne several children. The influence

FREQUENCY OF PREMATURE BIRTHS, AND NEONATAL MORTALITY FOR FULL-TERM AND PREMATURE INFANTS BY AGE OF MOTHER, NEW YORK STATE (EXCLUSIVE OF NEW YORK CITY), 1936.

Age of mother.	Frequency of premature births.*	Neonatal mortality-rates.*		
		Premature births.	Full-term births.	Full-term, single, and legitimate births.
Under 20 ..	60·5	369·4	15·5	14·1
20–23 ..	43·2	359·0	13·7	13·3
24–27 ..	36·4	397·8	13·5	13·2
28–31 ..	38·7	375·6	14·9	14·3
32–35 ..	42·0	428·6	15·7	16·0
36–39 ..	47·8	425·7	19·2	18·6
40 and over	56·3	430·2	25·3	23·6

* Per 1000 live births in each specified group.

of age of mother upon neonatal mortality is still apparent when "order of birth" is held constant. Even more astonishing and quite unexpected is the discovery that the age of the father exerts almost as great an influence on the welfare of the offspring as does the age of mother. And this again is no spurious correlation due to the correlation between age of husband and wife, for when age of mother is held constant the variation of neonatal mortality with age of father is still conspicuous. The mortality-rate starts high for very young fathers, drops to a minimum for fathers in the age-group 25–29, and thereafter rises with the father's age.

MEDICINE AND THE LAW

New Public Health Act Offence

ON Jan. 5th Harry Lovell, a rag-and-bone collector, was fined 40*s.* at Birmingham for infringing Section 154 of the Public Health Act of 1936, a new law of which he was probably unaware. He had distributed fountain-pens and a notebook to children under 14, after bartering for the articles which he collects. This was said to be the first prosecution of its kind.

The former law on the subject was to be found in Section 73 of the 1925 Act. It forbade a collector or dealer in rags or bones or similar articles to sell or distribute "any article of food or any balloon or other toy" from a cart, barrow, or other vehicle employed for the collection of rags and bones, &c., or from any shop or premises used in connexion with that kind of business. The purpose of this provision was obvious; its attempt to prevent the spread of vermin or infection was extended in some places by local legislation. The Local Government and Public Health Consolidation Committee, when framing the clause which became the present Section 154 of the 1936 Act, was made aware of difficulties encountered in enforcing Section 73 of the 1925 Act. Summonses against rag-and-bone dealers were sometimes dismissed because the dealer produced his *quid pro quo* (balloon, toy, or what not) from a bag or stock carried in his hand and not kept on the cart or barrow. Secondly, the words "any balloon or other toy" fostered forensic uncertainty. There is a principle of statutory construction known as the *ejusdem generis* rule. If a general word follows particular and specific words of the same nature as itself, it takes its meaning from those words and is deemed restricted to the same genus as those words. Thus the Prison Act forbade the conveyance into prison (with intent to facilitate the escape of a prisoner) of "any mask, dress or other disguise or any letter or any other article or thing"; the words "any other article" were held to include a crow-bar. But in a New Zealand case, where a statute for suppressing offensive publications used the phrase "advertisements or other publications," it was held that a pamphlet sold only as an appendix to a bona-fide medical work did not come within the phrase. Magistrate's courts in England had been invited to decide whether the words "other toy" in the 1925 Act meant some other toy of the same nature as a balloon; did the words include pencils, notebooks, or children's jewelry? To avert these technical problems the committee framed the new law in such a way as to forbid the rag-and-bone man (1) to sell or deliver, whether gratuitously or not, any article of food or drink to any person, and (2) to sell or deliver, whether gratuitously or not, any article whatsoever to a child under 14.

Duty to Notify Puerperal Fever

At an inquest on the death of two women from puerperal fever in the Melrose Nursing Home at Sutton the findings of the coroner's jury criticised the matron, a medical practitioner, and the local authority's deputy inspector of midwives and nursing-homes. The deaths were those of Mrs. Hodges and Mrs. Peskett. The pathologist who conducted the post-mortem examinations stated that he found septicæmia due to hæmolytic streptococcus. Twin daughters had been born to Mrs. Hodges in the home; both died, one from infection contracted in the home. An inquest had been held upon this death also; evidence was given that the two babies came in contact with the mother quite frequently.

At the inquiry into the two women's death it appeared that a Mrs. Skinner who showed signs of puerperal fever, was isolated and removed from the nursing-home to Queen Charlotte's Hospital. Mrs. Peskett, it was said, would also have been removed if her condition had not been too dangerous. A third patient, Mrs. Hodges, was admitted to the ward in the nursing-home which Mrs. Peskett had vacated, but was not placed in the same bed. The medical attendant of Mrs. Hodges notified her case when her temperature rose above 100° F. on Dec. 1st. He had not been aware of Mrs. Skinner's removal and he made no inquiries whether the nurses were infected, though he did inquire whether they were going to be examined. The medical attendant of Mrs. Peskett did not notify the case of his patient. He did not pass on to the matron any suspicion he entertained which would cause her to warn other patients against entering the home. The witness told the coroner that the matron informed him that she had not notified Mrs. Peskett's infection because she had not been in physical contact with her. The matron, in her evidence at the inquest, said that she asked Mrs. Peskett's doctor to notify the case because of the patient's high temperature; he replied that this was unnecessary but he would do so if she wished. She herself had not notified the case because the doctor did not do so; she did not report that she had been in contact with Mrs. Peskett because for nearly a month she had been off duty through neuritis and had not been in actual contact. Pressed by the coroner to explain why she had received Mrs. Hodges into the home on the day after isolating Mrs. Peskett, the matron answered that Mrs. Peskett's temperature might not have had a puerperal cause;

as the doctor had not suggested it, it was not for her to think it had. Dr. John Beattie, who is available under agreement with the Surrey county council for consultation in cases of childbirth in his prescribed area, had visited the home on Nov. 28th at the request of Mrs. Skinner's medical attendant. He later saw Mrs. Hodges in the same ward. He gave evidence that he would agree with the pathologist's findings as to death; in the case of Mrs. Peskett there was an intermittent temperature of over 100° F. for eight days, which would be strong evidence of puerperal fever. He saw no signs in the ward of masks or gloves; droplet infection was common and masks ought to be the rule. There would be no excuse for the nurse who attended Mrs. Peskett to attend another patient at the same time. Dr. F. Langford, of the Surrey county council health department, said that the case-books of the home complied with the regulations. He visited the home on Nov. 30th when Mrs. Hodges and Mrs. Peskett were still there. He then asked the matron why she had not notified her contact with a suspicious case. During his visit on Nov. 30th he took swabs (nose and throat) of the staff. The matron was negative, the two nurses were positive, and a sister was negative as to the nose and doubtful as to the throat. He advised the matron that it would be very dangerous to admit anyone else for the present. Discussing the possible origin of the infection, he ascertained that a Mrs. Willard had been admitted on Nov. 8th who had a temperature of 101° F. on Nov. 16th and of 100° on the 18th.

The jury returned a verdict of "death by misadventure" in both cases. A rider was added which criticised the matron for having admitted Mrs. Hodges when infection was suspected. Another rider criticised Mrs. Peskett's medical attendant for failure to take adequate post-natal care.

AN INTERNATIONAL REVIEW OF INFECTIOUS DISEASES

THE report of the October session of the permanent committee of the Office International d'Hygiène publique contains much valuable information about the major epidemic and endemic diseases and the steps being taken to control them. The following selection gives some idea of the work of which the committee takes note.

YELLOW FEVER

In the first nine months of 1937 yellow fever has been rather more frequent in Africa than in the immediately preceding periods. Small epidemics occurred at Accra and Mepom (Gold Coast) and single cases were notified at Brazzaville and Bangui in French Equatorial Africa, both of which are areas in which the disease had not previously been observed. In South America it was notified from Colombia and from Peru, the latter being the first notification for a long time, as well as from the ptates of Matto Grosso, Minas Geraes, and Sao Saulo in Brazil. In the Anglo-Egyptian Sudan fresh work has confirmed the high proportion of sera positive to the mouse-protection test, the proportion reaching 70–80 per cent. in the region of the Nuba mountains. It would seem that yellow fever existed there in 1936 in the neighbourhood of Kau: a large number of sera of children under ten years old were positive in 1937, whereas in 1935 all sera of children of the same age had been negative. That of a European doctor who had lived in the area in 1936 was negative before and positive after his stay; in the interval between these two examinations he had had a slight attack of fever lasting 4–5 days.

Virus attenuated by tissue-culture, without the addition of immune serum, has been used with success in England on more than 600 persons with no serious reactions. In North and South America the Rockefeller Foundation uses a virus grown on tissue from chick embryos from which the head and spinal cord have been removed. This has been used without immune serum on more than 6000 persons with a proportion of 5–8 per cent. of slight reactions on the sixth or seventh day after vaccination. Two to six months after vaccination 51 out of 2200 persons vaccinated by various methods showed jaundice, as to the causation of which various explanations were advanced.

TYPHUS FEVER

Although fairly common in Czechoslovakia between 1920 and 1923, typhus has now almost disappeared from that country except in the mountainous region of the north of Russian Sub-Carpathia, into which it is thought to be introduced from Poland by smugglers. The local population is comparatively immune but persons coming from other parts of Czechoslovakia are much more severely attacked. Weigl's vaccine has been used since 1934 for persons particularly exposed to infection; no case of the disease has occurred up to date in 740 persons vaccinated.

In Morocco there were three serious outbreaks after following the bad harvest of 1936—i.e., near Marrakesch, at Fez, and at Casablanca. Blanc's living vaccine attenuated with bile was used to inoculate the great majority of the natives in the threatened areas: 44,655, 2962, and 146,283 persons were vaccinated in the above three areas respectively. This mass vaccination was followed by a sudden fall in the epidemic curve, even although no other prophylactic measures such as delousing were employed. According to carefully controlled mass observations, the total percentage of reactions was 0·61 among the natives and 13 among Europeans.

In Tunisia endemic typhus has persisted in a few areas and broke out between November, 1936, and June, 1937, with unusual virulence. The results of the classical methods of prophylaxis having therefore been judged insufficient, it was decided to employ, in addition to isolation of infected persons and delousing, vaccination with Laigret's vaccine—i.e., living murine virus incorporated in egg-yolk. This can be dried and suspended in oil at the time of inoculation and can therefore be sent considerable distances and used by doctors on the spot. A total of 3601 vaccinations were made in Tunisia in 1935–36, 27,941 in 1936–37, and it is hoped to raise the figure to 150,000 for 1937–38. In vaccinated subjects there were 32 cases of typhus with no deaths, but 27 of these fell ill during the first fortnight after inoculation. The incidence of typhus in the months following inoculation was 0·9 per 1000 in the vaccinated and 11 per 1000 in the non-vaccinated of the same communities. Three cases of murine typhus, with a rash but of an ambulatory type, were produced by vaccination.

SMALL-POX AND VACCINATION

A total of 5 cases of post-vaccinal encephalitis were notified in Great Britain in 1936 and 3 cases in the first eight months of 1937; 5 of these 8 were fatal: 4 of the cases were in young infants, the others were primary vaccinations in children of ten to fifteen years. In Germany 5 cases were confirmed in 1936, 2 in children of three months and one year, the remainder in children of three and four years old. In Italy 5 cases occurred in 1936 and 6 in 1937 out of 1,104,520 primary vaccinations and 1,205,971 revaccinations in 1936 and the spring of 1937. All the cases followed primary vaccinations and 6 occurred in children of ten to thirteen months, while 5 were in children between two and eight years old. In Sweden, during the period 1924–36, 54 cases were notified (43 primary vaccinations and 11 revaccinations), of which 11 occurred in the last four years. No case was noted in the first year of life; 6 were in children between one and two years old. The

highest proportion of cases to vaccinations appears to occur in the age-period 5–10 years. Cases are scattered throughout the country and their frequency does not appear to be influenced by epidemics of poliomyelitis.

POLIOMYELITIS

Studies of several epidemics in Sweden show that poliomyelitis appears in several areas of a district at the same time and may be presumed therefore to depend on some external factor. Rainfall or thaws seem to occur some 10–20 days before each outbreak and cases appear first in the country and then spread to the towns, possibly through unpasteurised milk. Cases of reinfection have been noted and specific immunity does not appear to be important. In Italy, on the other hand, multiple cases in families or schools are almost never observed, nor is there a relation to the rainfall. In Switzerland, as compared with an average annual incidence of 300, 1269 cases occurred in 1936, and 1088 in the first ten months of 1937. Family cases are very rare but become commoner as an epidemic progresses.

SOME OTHER DISEASES

Scarlet fever.—In the United States scarlet fever continues to be mild in character. The case-incidence is 200 per 100,000 population and the fatality-rate is about 1 per cent. Active immunisation with Dick toxin makes little progress, except among those specially exposed to risk, such as nurses.

Bacillary dysentery.—In the Netherlands Shiga-Kruse bacillary dysentery has disappeared since the beginning of the century but small epidemics are fairly often observed, due to Flexner's or the Y bacillus, and particularly from Sonne's bacillus. Cases are mild but deaths may occur in children and old persons. The principal prophylactic measure is the discovery and elimination of carriers among food-handlers.

Tularæmia.—No further human cases of this disease have occurred in Czechoslovakia since the 1936 epidemic, but in the autumn of 1937 infected hares were found in two of the ten districts affected in 1936. Three human cases have been diagnosed in Germany, in 1931, 1933, and 1936, but the infection has not been discovered up to date in hares and rabbits. In Sweden 96 human cases were notified last summer, for the most part in the district of Bollnaes in central Sweden—the same area as was affected in 1934. The 3 first cases were caused by a hare found dead in the forest and an insect vector is suggested as the mode of infection for the remainder.

The representative of the United Kingdom on the committee is Dr. M. T. Morgan, of the Ministry of Health. Others present at the October meeting included Sir John Megaw, Sir Thomas Stanton, Colonel S. P. James, Dr. H. B. Jeffs, Dr. P. G. Stock, and Mr. O. F. H. Atkey.

GRAINS AND SCRUPLES

Under this heading appear week by week the unfettered thoughts of doctors in various occupations. Each contributor is responsible for the section for a month; his name can be seen later in the half-yearly index

FROM A BACTERIOLOGICAL BACK-NUMBER

III

I HAVE recently retired prematurely from all active work and among the melancholy jobs which have arisen therefrom has been the weeding out of all the books that I have accumulated during my professional life. Long ago my household had issued the ultimatum that the house simply would not, and could not, hold any more of my books, so I was reduced to the subterfuge of transferring all my medical ones to my laboratory. I don't pretend that I always made an honest and conscientious use of the laboratory accommodation. In fact I was liable to draw the line rather arbitrarily and treat all books which were unsuitable for shelves exposed to the impertinent curiosity of a young family as *ipso facto* medical. Thus the Decameron and the "Loose Songs and Doggerels" of the Percy Folio came to find a place in my medical library. During the height of the Christmas deliveries the books reached me packed in strong wooden boxes. The fact that the boxes were superscribed with such famed and seasonable names as Haig and Haig and Johnnie Walker inspired the otherwise heavy work of unlading, and the van-men pocketed their tip wishing me a very merry Christmas with a twinkle in the eye and just a hint of caution in the voice. When I set about unpacking I was shocked to find that the first box was packed with culture tubes fully loaded with bacteria. My old laboratory assistant had touchingly thought that it would be a consolation to me in my exile to have this *hortus siccus* from the old life. I don't know exactly what he expected me to do with it. Bacteria are as awkward a domestic problem as discarded safety-razor blades; modern dustmen simply won't stand for them and you can't very well put them in plum puddings. So at present this formidable collection stands at the foot of my bed awaiting the services of an expert convoy to the nearest autoclave which unfortunately is several miles away.

It was not quite without emotion that I gingerly handled perhaps for the last time some of the vintage strains of those elusive little creatures to whose consequences and inconsequences I had devoted so much of my life. It was sad to say farewell to K.L.B. Park-Williams 8, that most pampered of the Lilliputian tribe. It had made me many a brave gallon of toxin though at times it was as fractious as a prima donna. I remembered almost affectionately how I had once run a needle charged with it into my finger and as I was Schick-negative it produced a nice little pure-culture abscess, teaching me that the diphtheria bacillus could still lash out with its tail after its teeth were drawn. Then there was B C G and Typhoid Rawling, not to speak of a collection of very anæmic-looking *B. mesentericus niger*.

On the whole bacteria are strange and unsuitable bed-fellows and definitely unadapted to the country life, and I shall share the relief of the rest of the household when my collection finds its last resting-place in the disinfector.

* * *

When I came to going through the books the task became progressively more depressing. It was almost like homicide to throw to the flames these old once-treasured companions. I wished for some young and docile disciple to whom I could have passed them on; but could I find any present-day medical student who has time to read Trousseau? I paid a penny a volume for the Clinical Lectures when I was myself a student and never got better value for money, for I never learnt more from any medical work. Then there were the "Memoirs on Diphtheria" of Trousseau's great predecessor, Bretonneau, also in the dingy brown covers of the New Sydenham Society's edition, and not to be discarded without a pang, Murchison on Fevers, Graves's Clinical Lectures, Snow on Cholera, Hilton's "Rest and Pain,"

Gee's "Auscultation and Percussion," and Rickets on Small-pox—all hardly less worth reading than on the day of their publication and an invaluable corrective to the scrappy and heated-up fare of our tin-can age. I next unearthed that questionable but interesting classic, "The Extant Works of Aristotle"—I mean that uncanonical collection which for two or three centuries was the main guide or misguide of the ordinary man in matters of sex. I picked my copy off a Charing Cross-road bookstall. It was bound in a brown-paper cover and labelled "The Toilers of the Sea" by Nat Gould. I'm afraid I "bought" it in more senses than one. The old loose cover has gone, otherwise I should have presented the copy to a Victorian museum as an article of bigotry or virtue or both. And among the rest I found that greatest of all literature written by a medical man. It was labelled the "Works of François Rabelais, M.D." Strange that the addition of those two sober letters should give the necessary touch of pruriency to the title. It is a cheap ill-printed edition which I bought from a bookseller who, thinking he had fathomed my tastes, persuasively tried to press on me also a copy of Paul de Koch's "Devil on Two Sticks." It proved, however, to be the genuine and complete "Urquhart and Motteux" and I have, in fact, never possessed any more noble edition of this strange concoction of diamonds and dung. Though the world is so morbidly curious about him, Rabelais is probably one of the least read of the great authors of the world. He is essentially a "medical" classic and I doubt whether any but doctors can really stomach him. But he is not easy reading to anybody and to follow the wild flights of that reckless and teeming brain is calculated to make even the strongest head reel. Nothing to use his own expression, was "too hot or too heavy" for him. Listen to this profitable advice given to the good Pantagruel by his father the wise Gargantua:

"Now, in the matter of the knowledge of the works of nature, I would have thee to study that exactly; that so there be no sea, river, nor fountain, of which thou does not know the fishes; all the fowls of the air; all the several sorts of shrubs and trees, whether in forest or orchards; all the sorts of herbs and flowers that grow upon the ground; all the various metals that are hid within the bowels of the earth; together with all the diversity of precious stones that are to be seen in the orient and south parts of the world. Let nothing of all these be hidden from thee. Then fail not most carefully to peruse the books of the Greek, Arabian and Latin physicians, not despising the Talmudists and Cabalists; and by frequent anatomies get thee the perfect knowledge of that other world, called the microcosm, which is man. And at some of the hours during the day apply the mind to the study of the Holy Scriptures; first in Greek, the New Testament, with the epistles of the Apostles; and then the Old Testament in Hebrew. In brief, let me see thee an abyss and bottomless pit of knowledge; for from henceforward, as thou growest great and becomest a man, thou must part from this tranquillity and rest of study, thou must learn chivalry, warfare, and the exercises of the field, the better thereby to defend my house and our friends, and to succour and protect them at all their needs against the invasion and assaults of evildoers."

Medical education is here defined, but with what a grand sweep!

I think the works of François Rabelais, M.D., must be saved from the fire. I may have done with medicine and with bacteria and bacteriology but I still have to live, and in a somewhat difficult and depressing world. I may have further need of the doctor-curé of Meudon. But perhaps I had better rebind him in brown paper and label him "Cocktails and How to Mix Them" by Dean Inge or something equally misleading.

PROTECTION OF EYESIGHT

In connexion with the fifteenth International Ophthalmological Congress in Cairo (briefly reported in our last issue) meetings were held by the International Organisation against Trachoma and the International Association for the Prevention of Blindness.

To the former body, over which Mr. A. F. MacCallan (London) presided, Dr. Rowland P. Wilson, director of the ophthalmic memorial laboratory at Giza, gave an address on the pathology of trachoma in which he said that the theory that it has a bacterial origin has now been abandoned, and attention is concentrated mainly on its resemblance to diseases caused by known viruses. In Egypt 90 per cent. of the population is still infected with trachoma, and the question of reducing this permanent scourge is closely connected with the social conditions prevailing. The excellent work done at present in Egyptian hospitals can do no more than diminish complications resulting from trachoma and thus reduce the incidence of blindness.

Dr. A. Bailliart (Paris) presided over the International Association for Prevention of Blindness, and Mr. MacCallan discussed the measures adopted in Egypt. The ophthalmic campaign inaugurated 34 years ago, and especially the establishment of travelling ophthalmic hospitals, has, he said, reduced the incidence of blindness among patients attending ophthalmic hospitals by a third. Dr. F. Park Lewis (New York) spoke on the responsibility of the ophthalmologist in the conservation of sight. The prevention of blindness is as worthy of consideration by ophthalmologists as either research or surgical care. The blindness that comes from negligence, from ignorance, from indifference and the crudities of living and its mismanagement is universal. The proportion of eyes that are lost and might be saved by preventive measures is greater than the proportion in which sight is restored by science and by surgical skill, and the results are better.

Mr. Joseph Minton (London) read a paper on prevention of industrial eye injuries. An investigation had been carried out by him at the Royal Eye Hospital, London, where over 7000 patients attend every year suffering from such injuries. The present high numbers are due to (1) the non-provision of safety measures by employers and (2) the indifference of workmen to the risks entailed in their work and their neglect of the means of prevention supplied to them. Mr. Minton has organised a museum of preventive measures in the out-patient rooms at the Royal Eye Hospital; the patients thus have an opportunity of studying the preventive appliances and of choosing the one most suitable for their work. If other hospitals situated in industrial centres would take an interest in preventive work a diminution of eye injuries and consequent damage to eyesight would follow.

Mr. Lewis H. Carris (U.S.A.) read a paper on the rôle of the social worker in a campaign for preventing blindness. Educators, public health officials, nurses, industrialists, sanitary engineers, social workers, and all in any way concerned in human welfare must take their place in the attack upon this social enemy. The world at large must be taught the causes of blindness and of defective vision, the place of nutrition and hygiene in relation to the health of the eye, and the methods of safeguarding the eye against injury and harm, and of keeping it in a state of greatest possible usefulness.

CORRESPONDENCE

THE LONDON M.B. FINAL

To the Editor of THE LANCET

SIR,—The leading article in your issue of Jan. 8th renders an important service by bringing into the open a discussion which, as a note in your issue of Oct. 23rd (p. 972) revealed, had long pursued a subterranean course.

Any alteration in the final M.B., B.S. examination, in the direction of lowering its standard, must interest most particularly the medical graduates of London University. That they should have been kept in ignorance of a movement which was being actively discussed, as your note declared, "in academic and collegiate circles," was obviously unfair. Accordingly, the University of London Graduates' Association, in its covering letter supporting the candidature of Mr. Philip Mitchiner for the vacancy in the representation of medical graduates upon the senate occasioned by Mr. Walter Spencer's retirement, deemed it proper to acquaint the graduates with the main outline of the proposition for such an alteration of degree submitted to the senate of May, 1937, by the board of the faculty of medicine. You rightly say in your leader that Mr. Mitchiner's election, not yet completed but now assured, will "strengthen" the party opposed to the proposition emanating from the board of the faculty of medicine. It may interest your readers to know that within ten days of the issue of Mr. Mitchiner's address, in which he declared his opposition to the proposition, and six weeks before polling day, considerably more than one-third of the whole accessible electorate had signified in writing their intention to vote for Mr. Mitchiner and their desire to add their names to any list of his supporters. In an experience of 32 years of this particular electorate I have never known a more explosive expression of opinion than is represented by this response, which leaves no doubt of the resentment felt by graduates against the proposition.

The scheme submitted by the board of the faculty reproduces in all essential particulars the scheme brought before the Haldane Commission in June, 1911, by the two then presidents of the Royal Colleges. That scheme was to the effect that the final qualification of the Conjoint Board should excuse students who had passed the second M.B. examination from the examination in medicine, surgery, and midwifery of the final M.B., B.S. Lond. The Haldane report of 1913 contains an admirably reasoned argument, even more cogent now than then, for its rejection, which was made in the following pregnant final sentence: "We cannot recommend that the University should agree, perhaps irrevocably, to the examination of its students by examiners appointed by an external authority." The Commissioners did more than reject the proposition: they made provision against its revival. Their comment on this question resulted in the framing of the current statutes which expressly forbid the delegation of examining for the final M.B. to any outside body. Reversal of this statutory prohibition will probably require the consent of Parliament. If I know anything of that august assembly, after more than thirteen years of continuous membership of it, that consent, in view of the history of the question, will be difficult to obtain.

Of the 22 members of the faculty board 9, including its chairman, were not graduates of the University, and in the case of several of those who were graduates their loyalties lay with the Royal Colleges rather than with the University, facts which may explain the decision of the Board, reached by a moderate majority vote, to renew the suggestion made to the Haldane Commission by the presidents of the Royal Colleges 25 years ago, and decisively rejected by it. The pretext for this renewal—and I submit it is no more than a pretext—was found in a pronouncement by the court to the senate in December, 1936, communicated to the faculty board and forming the justification of its report to the senate. This communication, the sole authoritative statement upon this subject, is so important that I reproduce it.

> "In making their grants to schools, the Court's attention was again directed to the small number of students in medical schools who take a university degree, and the Court resolved that, in communicating to the medical schools the Court's decisions with regard to grant, those schools be informed that the Court have noted with regret that in comparison with the rest of the country the number of London students who achieve a university degree is very low; that they are aware that the causes contributing to this are many and complex; that the recent changes in the scheme of the examinations of the University may have the effect of making a degree more easily accessible; and that the Court hope that at the end of the next quinquennium the figures will show an improvement."

It will be discerned at once that the full text makes nonsense of the contention, so often advanced by those who are responsible for this movement, that the grant-giving body is exercising pressure upon the University which would justify so revolutionary and discreditable an expedient as the lowering of the standard of the M.B., B.S., and inviting the coöperation of an outside non-university and non-teaching body in so important an examination as that for the final degree in medicine. The faculty board's proposition is all the more surprising in that it completely ignores the revision of the medical curriculum for which the board was so largely responsible and upon which the court rightly based its confident expectation of improvement of the position, and further, in proposing that an agreement should be made exclusively between London University and the colleges, ignores the Universities of Oxford and Cambridge, which took a prominent part in that revision and which send so large a proportion of students to the London medical schools.

You touch lightly upon the consideration that the proposed coöperation would injure and perhaps extinguish the external degrees in medicine. The faculty board were informed this probable effect but pursued the project undeterred—and thereby revealed their abysmal ignorance of the history of a struggle which very nearly wrecked the University, and, if revived, would do it incalculable damage. You rightly point out that the acceptance by the schools of the Exchequer grant for university education imposes on the schools a contractual obligation to "maintain training of a university standard." The medical schools take nearly a quarter of the whole annual grant to London University. They are the only schools which admit a large proportion of non-university students to their classes. The Haldane Commissioners were so gravely dissatisfied with this position that they recommended the restriction of full university recognition to only three hospital schools which should admit undergraduates of Oxford, Cambridge, and London alone. A long-overdue reform was initiated by my own school, St. Mary's Hospital, just three years ago, when it

announced its intention to admit in the future only students entering for a university degree. That example has been followed to such an extent that at the present time, taking an average of the 12 medical schools, London undergraduates form nearly three-quarters of the whole student-body, the other quarter consisting of Oxford, Cambridge, and Conjoint students.

When one compares this position with that of 25 years ago when the Haldane report stated that "less than one-third of the students of London medical schools were undergraduates of London University," one may perhaps find the explanation for the present desperate anxiety of the Royal Colleges to safeguard the existence of conjoint students, thus threatened with exclusion from the London medical schools, by coming to an agreement with London University that might save the situation for themselves but at the cost of destroying the present high reputation of the M.B., B.S. Lond. The University is under no obligation to the Royal Colleges to justify that sacrifice. A former very famous principal of King's College, giving evidence before the Haldane Commission, said bluntly that the real reason for the small number of students in London medical schools who took a university degree was because "we have in the Conjoint Board the competition all through a student's career of a lower standard, and that has a very bad effect." The commission concurred with that explanation but none the less rejected the solution that would meet that competition by surrender of standard in the degree examination. All recent developments indicate that that competition, serious 25 years ago, is diminishing daily and is destined to disappear in the not remote future. The University is working out its own salvation and is rapidly reaching the aim you set before it, when "the normal sequence of an ordinary conscientious undergraduate's course of study" will be the award of the London degree, unimpaired in prestige and value.

I am, Sir, yours faithfully,

Wimpole-street, W., Jan. 8th. E. GRAHAM-LITTLE.

EARLY TREATMENT OF CATARACT

To the Editor of THE LANCET

SIR,—A recent report on cataract by the Prevention of Blindness Committee (66, Victoria-street, London, S.W.1) deserves more publicity than perhaps it has had. It comments among other things on the number of cataract cases that are not submitted to operation and it suggests possible remedies for this. I wish here especially to point out that early recognition of cataract, and continued close observation with tactful encouragement to the idea of operation, when this seems the best course, is of the utmost importance, and lies very often with the general practitioner. The report has mainly panel patients in mind.

We ophthalmic surgeons do not see more than a few of our cataracts early enough. It has been my unfortunate experience to have seen many at a stage when intra-ocular complications could not be detected, and operation is apt to be blamed for an unsuccessful result. One patient with glaucoma simplex without any lens opacities had been told by his doctor over a long period to "wait till the cataracts are ripe"; he was blind and nothing could be done. Only an expert can tell whether a cataract case has or has not glaucoma as well, and this is much easier at an early stage. Tobacco amblyopia along with cataract often presents difficulties and is apt to be missed. Too often these cases drift to an optician who wastes valuable time in trying many glasses while the optic nerves go on towards atrophy. One can only hope that if registration of opticians comes about it will be an offence not to refer all cases with early lens changes to an ophthalmic surgeon.

But the main object of this letter is to stir up the practitioner to a lively recollection of his responsibility in the matter of early cataract. The whole idea of "waiting till ripe" should be regarded as out of date. Modern methods of operation have largely done away with this period of long weary waiting in semi-darkness. The person to decide is the ophthalmic surgeon.

I am, Sir, yours faithfully,

Nottingham, Jan. 6th. A. CHRISTIE REID.

CHILD DISCIPLINE

To the Editor of THE LANCET

SIR,—In your issue of Jan. 1st Mr. A. S. Neill defends his fancy for rewarding the thieving child when it steals. Different people obtain the same effects on children by very different ways, and a child by no means necessarily needs the same methods of discipline in all the phases of its childhood. Thus the practical details of daily discipline depend too much on personality and are too variable to be discussed with profit, and I was careful in my address (Dec. 25th, 1937, p. 1469) to say that it was the principles underlying Mr. Neill's practice in this matter to which I objected. These he enunciates with some assurance: "juvenile stealing is emotional; it is symbolic; the stolen money or trinkets symbolise the love that the child feels he hasn't got. By giving him a reward (that is a love token) I give the child what he is looking for and he doesn't then require to steal. The other way—the appeal to reason—does not work, naturally, for the fundamental motive is always unconscious." This underlying principle, this explanation, this exposure of the "root cause," is to me no more than an unverifiable hypothesis in which I feel no obligation to believe. I see no reason to think that such unconscious motives are able to cause, still less to "require," specific acts on the part of a child; and why should they, out of all the motives in operation, be exalted to the key-position which Mr. Neill gives them? To my mind these psychological explanations of cause and effect are not impressive. Some of them have the appearance of containing an element of truishness, while others are so bizarre as to make a nightmare of life; but on the whole they leave out too much and are too facile to be acceptable.

John in Mr. Neill's narration is a case in point. He writes to his mother in pencil and gets back a letter remonstrating with him for his slovenliness. It is not difficult to suppose that in the conditioned atmosphere of Mr. Neill's school this chilly breath of criticism ruffles the boy's temper and he is ready for a row. He falls foul of the cook and develops his best form in the hope, it may be, of getting some appetizing love token off her. But Mr. Neill will have it that there is more to it than this, and that the boy was led ("required," I suppose,) to row the cook in particular as "the best mother substitute—the food-giver." I see no reason to take this on, especially as I have an uncomfortable feeling that, had the boy shown his irritability by kicking the dog or punching the head of his neighbour, some equally specious explanation would have been forthcoming. As I understand it John is aware of his mother's displeasure and this he interprets, none too

sportingly, to mean a lack of love on the part of his mother. There seems small need to invoke an unconscious motive, and still less to make it responsible for the precise action which the boy takes. I hold no brief for John, conscious or unconscious; but Mr. Neill's art portrays him to me as a boy who knows his way about his school without the assistance of any unconscious direction.

I cannot forbear to acknowledge the kindly insinuation of Mr. Neill's concluding paragraph. But he may be reassured: I inhabit, alas, no such world. I am, Sir, yours faithfully,

Harley-street, W., Jan. 7th. REGINALD MILLER.

ROYAL MEDICAL BENEVOLENT FUND

To the Editor of THE LANCET

SIR,—I am extremely grateful for the most generous response which has been made this year towards our Christmas gift fund. The amount subscribed, £979 3*s*. 8*d*., has enabled us to give a Christmas gift of 30*s*. to every one of our regular beneficiaries. My thanks are due to you, Sir, for bringing the appeal before your readers, and to all who responded we desire to express our very sincere thanks. The committee is equally indebted to the many secretaries of medical societies, panel committees, hospital staffs, and divisions and branches of the British Medical Association, who brought the Christmas gift appeal to the notice of their members and who arranged for collections to be made at meetings, or donations to be voted. No less than 115 cheques from the above-mentioned secretaries, amounting to £511 15*s*. 8*d*., have been received.

We are particularly impressed by the spirit of goodwill and friendship shown towards the work of the Royal Medical Benevolent Fund in the letters received from many contributors. Practically all "wish every success to the appeal," others refer to the object of the appeal as being "most deserving." It is clearly shown that it is the desire of members of the medical profession to extend sympathy and practical help, through the agency of the Royal Medical Benevolent Fund, to those members of the profession who are living in adversity. My committee is confident that it is the wish of all that the Fund should be worthy of the traditions of our great profession—to be able to give adequate help to those in need. Great advance has been made since the early days and much has been done. We look now to those who have not yet become annual subscribers to join in with us during 1938.

I wish all a happy New Year and I thank you for the generous coöperation extended by you and your readers to the Royal Medical Benevolent Fund.

I am, Sir, yours faithfully,

THOS. BARLOW,
President.

11, Chandos-street, Cavendish-square, W., Jan. 5th.

PS.—Extract from one of many letters of thanks from beneficiaries. The writer is a widow aged 63 suffering from asthma and bronchitis. "Will you please give my very sincere thanks to the committee for the generous and unlooked-for Christmas gift. It came as a most timely gift. A month ago my doctor ordered me to bed and said that the fire must be kept going night and day, so instead of a 2*s*. 8*d*. bag of coal I was able to get ¼ ton. I felt it was being very extravagant and yet on Christmas Eve after the light was turned out it was lovely to lie in bed and think of all the blessings one was enjoying through kindness of unlooked-for friends."

HIRE-PURCHASE RESEARCH

To the Editor of THE LANCET

SIR,—The council of the Medical Defence Union is somewhat disturbed at the continuing volume of requests it receives for advice and assistance from practitioners who have entered into contracts with a body known as the Consolidated World Research Society Ltd. In your columns on Feb. 6th last there appeared under the heading "hire-purchase research" an excellent little article on the activities of the society already named. That article set out in full the type of encyclopædia which is proffered by representatives of the company to members of the medical profession. It also drew attention to certain privileges which canvassers claimed to be invaluable to any practitioner engaged in active practice. For the sum of £19 10*s*. there is offered (*a*) perpetual illustrated quarterly extension service for a period of ten years, (*b*) binders for the quarterly extension service, and (*c*) membership of the Consolidated World Research Society Ltd. for ten years. The purchaser is required to make a declaration that he has entered into this contract solely upon the terms and conditions contained therein and irrespective of any representations made to him by the company's representatives. Apparently many practitioners sign these contracts without reading carefully the declaration they have to make and then subsequently they express dissatisfaction with the services rendered, particularly those dealing with medical research. Under this heading subscribers are informed that they may approach the research department of the company for information on medical subjects in which they have a professional interest. You, Sir, rightly drew attention to the privileges of membership and stated that so far as they depend upon the printed agreement they lacked both definition and value. I should like to quote the following extract from your article, for it may on this occasion be read by many practitioners who did not have an opportunity of reading it previously, and they may thus become aware of the need for exercising care when entering into a contract as described above:

> One, at least, of the medical subscribers has placed on record his experience of them. He signed a printed contract and paid a deposit of £1 10*s*., but, being dissatisfied with the representations made to him by the company's agent, he afterwards repudiated the agreement. The company issued a summons in the county court, claiming £18 as the balance due for purchase of the encyclopædia. The defendant doctor, having taken the advice of the Medical Defence Union, put in a defence on oath in which he gave his own version of the transaction as follows:—
>
> "I accepted the verbal offer of the representative of the plaintiffs . . . that, for a fee of £1 10*s*. down and ten subsequent yearly payments of £1 10*s*. each, the plaintiffs' society would answer any research questions by me up to a specified number per annum, and I made the first payment of £1 10*s*. to him accordingly. . . . I discovered I had been fraudulently deceived and misled by the plaintiffs' representative, and applied to the plaintiffs for a return of the £1 10*s*. paid by me. Any contract set up by the plaintiffs as made by me to purchase any encyclopædia or other books was obtained by fraud and wilful misrepresentation by the plaintiffs' representatives."

It must have been an uncomfortable reply for the Consolidated World Research Society Ltd. to receive. No commercial undertaking can relish so wholehearted an allegation that its agents are obtaining business for it by fraud. The defendant doctor, however, was not given the opportunity to repeat his story in court. A few days before the date fixed for hearing, notice to discontinue the claim was filed

on behalf of the society. If the society feels it has been unlucky in devising a system of enterprise which permits such allegations to be made, it will doubtless reconsider its method of doing business.

Furthermore, members of the Medical Defence Union are requested to communicate with its secretary before entering into any contract with the Consolidated World Research Society Ltd. At the same time it would be desirable for them to indicate the scope and character of any representations made by any canvasser employed by the above society when approaching him on its behalf.

I am, Sir, yours faithfully,

ROBERT FORBES,
49, Bedford-square, W.C., Jan. 7th. Secretary.

MELANOSIS COLI

To the Editor of THE LANCET

SIR,—Until recently melanosis coli was almost universally regarded as the direct result of constipation. In my paper in the *Guy's Hospital Reports* for October, 1937, I brought forward evidence, derived from the admirable clinical investigations of Dr. H. L. Bockus and his colleagues and from my own observations, to show that it was the artificial diarrhœa produced by aperients taken on account of constipation and not the constipation itself which caused melanosis. There is still insufficient chemical evidence to show with certainty whether the precursor of the melanin is a product of the decomposition of proteins in the semi-liquid contents of the colon, or whether, as Bockus believes, it is a constituent of anthracene laxatives, although such evidence as there is is certainly in favour of the former theory. In view of the fact that anthracene laxatives are nowadays habitually used far more frequently than any others it is natural that most patients with melanosis should have treated their constipation with these drugs, but the patient described by Williams, a woman of seventy-four, had for the last forty-three years of her life taken only a grain of calomel every night or on rare occasions a blue pill or grey powder.

The frequent association of melanosis with carcinoma of the colon affords, I think, conclusive evidence against Bockus's theory. Stewart and Hickman [1] found melanosis visible to the naked eye in 49 per cent. of 43 autopsies on cases of cancer of the colon compared with 13 per cent. of 284 non-cancer cases, all being over the age of forty. In a microscopically investigated series of 100 cases of cancer of the colon they found a still higher incidence—55 per cent. of melanosis visible to the naked eye and an additional 13 per cent. in which melanosis was found microscopically. In 50 out of 60 cases the melanosis was present only above, or was more marked above, the growth, in 9 it was the same above and below, and in only 1 was it more marked below. The stasis of semi-fluid fæces in the colon proximal to the carcinomatous obstruction must lead independently of the taking of aperients to excessive putrefaction and the production of excess of the substances which form melanin on absorption by the mucous membrane.

Dr. Bockus is thus incorrect in his statement that my evidence against his views was based upon two case reports; these were not quoted with this object at all, but in order to show that melanosis coli disappears when the artificial diarrhœa caused by aperients ceases.—I am, Sir, yours faithfully,

ARTHUR F. HURST.
New Lodge Clinic, Windsor Forest, Jan. 8th.

[1] Stewart, M. J., and Hickman, E. M., *J. Path. Bact.* 1931, **34**, 61.

WHO PAYS THE DOCTOR CALLED TO AN ACCIDENT?

To the Editor of THE LANCET

SIR,—I was a little surprised, when reading your annotation in your issue of Jan. 1st to see that you made no mention of Sections 16 and 17 of the Road Traffic Act, 1934. In order to make exactly the provision for which you pointed out the need, Parliament enacted that when treatment or an examination is immediately required as a result of a motor accident and is given by a registered practitioner, the person using the car must pay the doctor a fee and expenses. If more than one doctor gives emergency treatment, the car-user need only pay the first to give it. The fee is 12*s.* 6*d.* for each person treated, and the expenses are 6*d.* per mile coming and going for any journey the doctor has to make over two miles from where he is called. If treatment is first given in hospital, the fee is payable to the hospital.

The doctor may claim his fee by word of mouth at the time, or may request it in writing, but he must serve his request on the user of the car within seven days, signing it and stating his name and address, the circumstances in which he gave the treatment, and that it was first given by him. If the user refuses to pay, the doctor can recover the fee as an ordinary debt. The chief officer of police must give the doctor any information he can about the identification marks of the car or the name and address of the user.

It is possible that doctors and hospitals have not made much use of these provisions, and I cannot recollect seeing any mention of them in any legal or medical paper since the Act was passed. The question of who is using the motor-car at the particular time may raise difficulty, and few doctors have time to take steps to trace an elusive user or sue an obstinate one. The sections, of course, apply only to motor-car accidents. I should be very interested to hear any experiences about their working in practice.

I am, Sir, yours faithfully,

London, E.C.4, Jan. 11th. D. HARCOURT KITCHIN.

INDEX TO "THE LANCET," VOL. II, 1937

THE Index and Title-page to Vol. II, 1937, which was completed with the issue of Dec. 25th, is now in preparation. A copy will be sent gratis to subscribers on receipt of a post card addressed to the Manager of THE LANCET, 7, Adam-street, Adelphi, W.C.2. Subscribers who have not already indicated their desire to receive Indexes regularly as published should do so now.

ROYAL MEDICAL BENEVOLENT FUND.—At a recent meeting of the committee eight annuities were awarded, and forty-five grants, amounting to £1204, were voted to beneficiaries. Among thirteen new applicants the following particulars are given of two cases:—

M.R.C.S. Eng., aged 70. Married. Invalided out of R.A.F. with glaucoma in 1926; has not been in practice since but has been able to earn a very small and precarious living by writing medical articles in the press. This source of income is ended owing to blindness. His wife, aged 58, has had several attacks of cholecystitis due to gall-stones and may have to be operated on in the near future. Private income nil. Fund voted £160.

Daughter, aged 60, of F.R.C.S. Eng. For 29 years has worked as a matron at two private schools, but had to give up her work in 1933 owing to phlebitis and varicose veins. She has been living on her savings which are now reduced to £30. She has no relations who can help her. Fund voted £36 and is obtaining assistance from other charities for this most deserving case.

Subscriptions and donations are urgently needed, and cheques may be made payable to the hon. treasurer, Royal Medical Benevolent Fund, 11, Chandos-street, Cavendish-square, London, W.1.

CROYDON TYPHOID INQUIRY

(*Concluded from p. 113*)

Evidence of the Consulting Analyst

IN the course of his examination by Sir Walter Monckton (counsel for the Croydon Corporation) on Jan. 4th, Dr. E. V. Suckling said it was improbable though not impossible that infection reached the well from the gathering ground, and that the most likely source of pollution there was the latrine trench near the heading. Some unidentified carrier could not be excluded, but the most likely cause of the outbreak was a carrier in the well. Carriers may be fæcal or urinary, the fæcal being the commoner, and a person may be a carrier only intermittently. To acquit an established carrier even five or six examinations at intervals of six days would not be conclusive, and more would certainly be needed with a urinary carrier.

DR. SUCKLING EXAMINED BY COUNSEL FOR THE TYPHOID OUTBREAK COMMITTEE

Mr. Gates asked how infection could have been introduced by the workman "Case A," seeing that he was almost certainly not a urinary carrier and it was impossible for anyone to have excreted at the bottom of the well. Dr. Suckling said that, in spite of the examinations that were made, this man may have excreted typhoid bacilli at some time in his urine, as more than half of carriers of the fæcal type had been found to do intermittently. The presence of large numbers of *B. coli* in the water could not be due to the carrier, and showed that sewage contamination was present also. The sewage of large communities (such as Edinburgh) usually contained small numbers of typhoid bacilli. "Case A" was, he thought, a chronic carrier and had been one for many years. Pressed by counsel he said it was possible that "Case A," who had had typhoid during the war and was therefore immune, might have become a carrier through drinking typhoid bacilli in the well water.

Asked by Mr. Gates how often he would advise sampling a water-supply, Dr. Suckling said that this depended upon many factors, but he did not agree with the practice of going from April to September without a sample. He agreed that the analyses of the Croydon supply did not tally with current procedure; there were no counts of agar plates and no attempt to define the type of coliform bacillus present. He did not know what was meant by the term "liver-milk" which occurred frequently in the analyses. The main difficulty in analyses of water was their interpretation and the analyses had not been seen by a bacteriologist of special experience. They were drawn up more from a chemical than a bacteriological point of view, though the latter was the more important as regards health.

Dr. Suckling agreed that a standard of not more than 100 colonies per c.cm. was aimed at in a water-supply, though that figure by itself would not greatly affect his opinion of a sample. Mr. Gates drew his attention to the report on the sample taken on Oct. 29th, in which there were 230 colonies per c.cm. and coliform bacilli were present in 10 c.cm.

Mr. Gates: Do you think any responsible analyst ought to make the comment on that: "This water is a good water, but I think the chlorinator should be used as a precaution"?—I do not agree with the description of it as a good water.

If you had received that sample, and were reporting on it, what would you have put at the bottom of the analysis?—I should have regarded it as polluted water.

You would not have recommended chlorination as a precaution, would you? You would have said "It must not be put into supply until chlorinated"?—Certainly.

In reply to Mr. Gourley, Dr. Suckling said that to render such a water potable he would have advised chlorination at from 0·15 to 0·2 per million. A dose of 0·05 per million was too small. In a neighbouring water-supply with which he was familiar, the water was first chlorinated at 0·15 to 0·2 part per million and then softened by Clark's process. Rapid filters could not be relied upon to remove bacteria.

Dr. Suckling thought that where an alternative supply was available a well should be put out of service while work was going on underground, but in this case the Stroud Green well would not have been adequate to maintain the supply. Since Nov. 4th, when the Addington well was shut off, the high-level supply had been supplemented by boosting from the low level, necessitating the installation of special pumps and the taking of more water from the Metropolitan Water Board. He agreed that no carrier should be employed by a water undertaking, and that it would be prudent before taking a man on to inquire whether he had had typhoid, and if he had, not to employ him until tests had shown that he was not a carrier.

Later, in reply to Mr. Gates, Dr. Suckling said that in his opinion early warning to the public and inoculations might have been of value in preventing secondary cases. On the following day (in reply to Mr. Lyons) he said that where there were known to be even a few cases of typhoid in a neighbourhood every person with vague illness should be suspected of having it until it had been excluded. It would be of no special assistance in diagnosis to know that a patient had been drinking water which was contaminated with typhoid.

DR. SUCKLING RE-EXAMINED BY COUNSEL FOR DR. HOLDEN

Replying to Mr. Sandlands, Dr. Suckling agreed that at the beginning of an outbreak it was impossible to say whether the cause was more likely to be milk or water.

Mr. Sandlands: From August 20th, 1936, until and including Sept. 29th, 1937, the Addington distribution main is examined no less than twelve times. . . . On no occasion is there *B. coli* in 100 c.cm. McConkey, although in the analyses of the wells you find *B. coli* in 6 out of 19 in Stroud Green, and in 8 out of 18 in Addington well. Bearing that in mind . . . and knowing by what you are told from the borough engineer that chlorination is in progress at each of the four wells, you would be very slow to suspect water alone when you have four cases notified, and two more suspected in hospital?—On those assumptions, yes.

DR. SUCKLING RE-EXAMINED BY COUNSEL FOR THE CROYDON CORPORATION

Referring to the Bournemouth outbreak, where typhoid organisms in water infected milk, Dr. Suckling explained that whereas bacteria die in water they multiply in milk, so that a small infection of milk may give rise to a widespread epidemic.

On the question of carriers, Sir Walter Monckton quoted from a report of the Medical Research Council that 61 per cent. of chronic typhoid carriers in the German army had been found to excrete the organisms in both urine and fæces. Dr. Suckling agreed that a carrier working in a well, even though he obeyed instructions with regard to coming to the

surface to defæcate, might convey infection to the water by his hands or clothing.

The presence of *B. coli* in a water sample indicates, said Dr. Suckling, either sewage or manurial pollution, but does not differentiate between them. The latter is of no importance in causing typhoid. Water which contains *B. coli* in 0·1 c.cm. may be described as highly contaminated, though milk containing *B. coli* in 0·1 c.cm. would be a high-grade milk. Until recently water from chalk supplies enjoyed a high reputation for purity, passage through chalk being thought to provide filtration, but this is now known to be erroneous. It was impossible to lay down a strength of chlorine required to apply to all water-supplies, as the amount of chlorine which different waters absorb varies widely. Adequate purification at Addington could be obtained by filtration and chlorination at 0·4 part per million. Even if samples of water were taken two or three times daily there might still be a risk of infection reaching a supply. Where a well had a chequered history and there were potential sources of pollution the necessary steps by way of filtration and chlorination had to be taken.

In Dr. Suckling's opinion the fever hospital which had been at Addington during the war could have no possible bearing upon the present outbreak.

Mr. Lyons: If four or five men urinated in the same bucket and one of them was a carrier . . . might the spilling of a teaspoonful from this bucket into a water-supply infect a whole area?—Dr. Suckling: Yes, if it contained typhoid bacilli.

Dr. Holden Recalled

In answer to Sir Walter Monckton Dr. Holden said that he had made inquiries where there had been more than one case in a family, and, except in one instance, he thought that the infection was probably primary in all members of the family.

Sir Humphry Rolleston asked whether some examples of very long incubation periods may not be due to a primary attack passing unnoticed and a relapse being regarded as the first onset. Dr. Holden said that he had had one definite instance of this. Sir Humphry also asked Dr. Holden when he would say that the present cases became an epidemic. Dr. Holden said that he first began to be concerned on Oct. 30th when he knew of six cases which were all in different families and different parts of the town. It was then that he started very seriously to wonder what was going to happen.

In cross-examination by Mr. Lyons Dr. Holden said that he first discussed matters with Mr. Boast, the borough engineer, when he met him at Mr. Rimington's house on the afternoon of Oct. 31st. He did not know that Mr. Boast had telephoned to Mr. Brandram-Jones at Brighton that evening and found out that the water at Addington had not been chlorinated for the past month until Mr. Boast had called on him at about 10 P.M.

Mr. Lyons: And what did he say to you about this matter—"I have just telephoned to Mr. Jones and what do you think, this water has never been chlorinated for the last month"?—I cannot remember what he said now.

But you realise the prime importance of that communication, do you not?—We did realise the seriousness of that information, yes.

Mr. Lyons: This was the first information you had of what you will agree is a highly vital piece of news?—It was the first information I had.

Mr. Lyons: Which came, of course, as a complete thunderbolt to you?—It came as a surprise to me.

Mr. Lyons: Did you at once, when you got the communication, connect the position and say, "Well, here is the avenue that has to be approached"?—I did not.

Dr. Holden agreed that Mr. Rimington had written to him on Oct. 29th saying, "The milk supplied in all cases is not the same; shell-fish and watercress have not been partaken of; the only common thing appears to be water." The matter had not been allowed to rest; he was following up every source of information that he could find. On Oct. 31st Mr. Boast said that he had telephoned to Mr. Jones at Brighton and found out that chlorination at Addington had not been continuous. Dr. Holden had said that if that were the case, as a measure of precaution, he had better start again. It was not until Nov. 15th that he had learned that men had been working down the well.

Mr. Forbes asked Dr. Holden whether it had occurred to him to confer with the local medical and panel committee. Dr. Holden replied that he had had very little to do with this committee, and did not know what it constituted.

Asked by Mr. Sandlands whether as the result of the evidence that he had heard and the inquiries that he had made he could suggest the source of the present infection, Dr. Holden said he thought the source of the outbreak was water from the Addington well infected by the man who had been found to be a persistent carrier of the fæcal type working down the well. This did not necessarily imply any wilful misconduct on the part of the man, as the possibilities of accidents in an adit with a bucket, hurricane lamps, and so forth were numerous. If the normal chlorination had been in force it would have been sufficient to have killed any typhoid bacilli which had got into the well.

Evidence of the Town Clerk

In reply to Mr. Forbes, Mr. Ernest Taberner, the town clerk of Croydon, said that the corporation were in favour of the fullest coöperation between the local medical practitioners and the public health services. Asked whether the corporation were in favour of the coöption of doctors on the public health committee, Mr. Taberner said that the corporation in their standing orders had decided not to exercise their right of coöption except with regard to such statutory committees as the maternity and child welfare committee.

Mr. Gourley said it had been suggested that the M.O.H. should coöperate with one or other medical bodies in the town, but he took it that it would not be considered right that a matter of this sort should be handled by a committee. There must be one captain on the bridge, and the corporation would look to the M.O.H. to be the man on the bridge. Mr. Taberner replied that the ultimate responsibility must rest with the M.O.H. If a committee had been in existence he doubted whether it could have told him anything that he did not already know or could find out from a standard text-book.

Mr. Forbes said the suggestion now was not that the local doctors might have assisted the M.O.H. but that he might have assisted them.

This concluded the evidence called by the corporation.

Evidence on Behalf of the Typhoid Outbreak Committee

In his opening remarks Mr. Lyons said he had suggested that the M.O.H had circulated in his report a statement that the water was good and there was no cause for alarm, when he ought to have known that he should have added "This water is being sterilised because of the apprehensions I have." He wished to make it perfectly clear that there was

no suggestion that Dr. Holden had deliberately composed something that was false. He proposed to call evidence as to what happened at the meeting on Oct. 31st.

Mr. R. L. Moss, a resident in South Croydon, said he attended a meeting at a private house on Oct. 31st when the M.O.H., the borough engineer, and some 40 local residents were present. Mr. Moss had had some experience with typhoid in India. He had asked Dr. Holden whether he thought that water was a possible source of infection, and Dr. Holden had replied (to his amazement) that in his opinion it was inconceivable as the water had been tested. Dr. Holden had said that he had eliminated milk as a cause and was concentrating on food. Asked whether he had told local doctors about the outbreak, Dr. Holden had said that he had not notified them, nor told them about the symptoms to look out for, but that he thought it would be a very good thing to do. Mr. Moss had suggested that the inhabitants should be advised to boil their water, either by means of the B.B.C., the press, or cinemas, but Dr. Holden had opposed this as it would create a scare.

Sir William Willcox Examined

Sir William Willcox, senior medical adviser to the Home Office, examined by counsel for the Typhoid Outbreak Committee, said he had personally seen 16 of the Croydon typhoid cases at the London Fever Hospital, of which he was senior physician. He would regard the present outbreak as a serious one and the mortality-rate which looked like being about 15 per cent. as fairly high. In the Dardanelles and Mesopotamia typhoid was due to flies, dust, and such things, but in England the common causes were water and milk.

Mr. Lyons: Will you tell the tribunal what is your view upon the closest collaboration that should take place between the medical officer of the district and the local medical practitioners?—I think it is most important that one should have close contact and close liaison between the M.O.H., the municipal authorities, the general practitioners, and a group of doctors of the voluntary hospitals. . . . Now, I am not criticising the M.O.H. of Croydon (far be it from me to come here and do that) but I do criticise the machinery which exists throughout the whole country to-day. . . . There should be some ad-hoc committee representing the medical practitioners of the district which can advise the M.O.H. if called upon and which can be, in turn, helped by the M.O.H. . . . It is easy to say that the general practitioner does not know much about public health. Perhaps he may not, but he is the first one to establish contact with the patient and the patient's family; the first one who can put in simple language to the public the dangers they are exposed to and the precautions they should take.

Sir William said he had seen the circulars sent to local doctors on Nov. 1st and 4th, but he would like to see closer contact than that between medical practitioners and the health authorities. A number of points might usefully be discussed by the M.O.H. with local doctors at the beginning of an outbreak such as this: the advisability of boiling water, the earliest signs to look out for, and blood tests. Leucopenia was a very suggestive sign and was present in the first two or three days. A positive blood culture might be obtained on the third or fourth day. These were matters which might have been discussed by the M.O.H., who should act as a sort of supreme adviser to local doctors.

Asked about prophylactic inoculation, Sir William said that the advisability of this must be decided on each individual case. He would not advise it during the incubation period, but if one could be certain that a person had been free from any chance of infection for three weeks, one could give prophylactic inoculation with advantage.

If the public were instructed in the way in which typhoid infection was spread you would not have alarms and scares about the danger of shopping in or visiting the district.

Sir Walter Monckton (counsel for the Croydon Corporation) read an extract from the report of Judge Cotes-Preedy on the typhoid outbreak at Bournemouth and Poole:

"It is common knowledge that sporadic cases of typhoid fever frequently occur all over this country, and in my view the officials of the boroughs would have acted unwisely if, directly cases of illness suspected to be paratyphoid or perhaps typhoid had appeared, they had sponsored an alarmist press announcement of an existing typhoid epidemic . . . had such a pronouncement been made before the precise nature and source of the disease had been determined, a hasty flight of people away from the district carrying the disease to all parts of the country might have occurred, thereby preventing the localisation of the trouble."

Sir William agreed that this report would be known to the M.O.H.

Sir Walter Monckton suggested that if the M.O.H. wanted advice he should turn to the Ministry of Health.—Sir William agreed and said that from the scientific point of view the diagnosis in this case of the cause of the outbreak was very quickly made. His criticism was that there was rather a lack of liaison between the general practitioners and the M.O.H. in dealing with the problem which had eventuated from the infection. The letter sent to doctors on Nov. 3rd telling them that a few cases of typhoid fever had occurred in the district was all right as far as it went.

In reply to Mr. Sandlands, Sir William said this epidemic was rather sprung upon Dr. Holden and perhaps he did not realise the help that an ad-hoc committee of local practitioners might have given him.

Later, in answer to a question by Sir Humphry Rolleston, Sir William said that from reading the evidence he favoured the view that the Addington well was infected by sewage in the area surrounding the well which had been contaminated by a carrier, the germs reaching the water by percolation through the chalk. He thought this more likely than that the infection should have been due to a carrier working in the well.

Sir Humphry also asked whether Sir William knew of any examples of a standing medical committee existing to advise the M.O.H.—Sir William said there were committees in Manchester and Liverpool and several other big cities. He did not suggest that power should be taken from the M.O.H. and given to the committees; the M.O.H. must remain at the head of public health matters, but no one man can advise on the many little difficult details which arise in an epidemic, and he thought that he might have been greatly helped, greatly supported, and greatly relieved in his responsibilities by having a strong ad-hoc committee to advise him.

(When the inquiry reopened after the luncheon adjournment Mr. Murphy pointed out that when he had asked Sir William Willcox for his opinion of the cause of the outbreak they had all assumed he knew that a carrier had been working in the well. Actually no evidence had so far been called to show that this man was a carrier, and Sir William's opinion was therefore given without that knowledge.)

Evidence of the Medical Officer from the Ministry of Health

Before Dr. E. T. Conybeare was called Mr. Murphy said he would carry out his examination himself, so that he would be nobody's witness. It would be as well to avoid questions of opinion as to where blame should be allotted as the impression might be created that Dr. Conybeare was expressing the judgment of the Ministry of Health.

Dr. Conybeare said he went to Croydon on Nov. 3rd to confer with Dr. Holden. He attended a meeting on his arrival at which the M.O.H. and the town clerk were present. After this Dr. Holden told him frankly that he was puzzled regarding the causation of the cases. A map was obtained from the borough engineer from which it appeared that eight of the nine cases were in areas apparently supplied by the high level system. He had been shown the recent analyses of the water and had suggested that samples should be taken from the two high level wells and sent to Dr. W. M. Scott at the Ministry. He was told that chlorination had been in force since Nov. 1st, purely as a precaution. The following day he returned to the Ministry and while he was there Dr. Scott rang up to report on the two samples he had been sent, which showed that the Addington well was heavily contaminated. He returned to Croydon and urged that the supply from the Addington well should be cut off and an extra point of chlorination introduced at the outlet from the reservoir. That afternoon, Nov. 4th, he visited the well and understood from the borough engineer that no fresh adits had been driven in it lately. On the 5th he visited two schools in the neighbourhood and inquired for an illness which often occurs within 72 hours of the ingestion of typhoid germs—namely, vomiting or diarrhœa. This may be due to the ingestion of colon bacilli or other sewage organism, but is sometimes useful in indicating when the infecting material has been consumed. In the two schools he obtained records of 19 such illnesses, of which 15 occurred between Oct. 19th and 28th. None of these cases had since developed typhoid. The information at the schools strongly supported the view that the infection was water-borne. The ramifications of the water-supply were so complicated that an engineering inspector from the Ministry had been sent down and Dr. Conybeare had visited all the wells and the reservoirs with him and Dr. E. L. Sturdee on Nov. 8th. On that day Dr. Scott had reported that he had been unable to isolate the typhoid bacillus from the water samples. He had advised that a special consultant should be called in by the corporation to advise on the water, and Dr. Suckling had arrived for this purpose on Nov. 9th. On Nov. 12th he had first heard from the engineering department of the Ministry that work had been going on in the Addington well for some time up to Oct. 28th. On the 13th he had obtained details of this work from Mr. Ellis, the mechanical superintendent, and the names of the 18 workmen engaged upon it. On Nov. 15th he had interviewed all these workmen and obtained a medical history and a sample of blood from each. As a result of the tests of these blood samples four men were sent to the Mayday Hospital during the following week for investigation of their urine and fæces. In one of these, "Case A," five specimens of fæces had been examined and all had proved positive for typhoid bacilli, but 12 specimens of urine had all been negative. None of the specimens from the other workmen had been definitely positive. On the 19th he had inquired into the question of watercress and had found that this could not possibly have entered into the causation. On Dec. 1st he had received bacteriological reports upon a large number of samples from the gathering ground, the Addington well, the reservoir and its neighbourhood and the supply distribution. None had shown typhoid bacilli.

Asked whether with the knowledge that he now possessed he could say that the probable cause of the infection was the known carrier in the well or some unknown carrier who might have polluted the gathering ground, Dr. Conybeare replied:

"I think that the coincidence between the dates on which this man was working in the well and the probable dates of infection in the majority of cases is no less remarkable than the coincidence between the distribution of the cases in the high level water-supply. On that ground I do regard it as the most likely probability that he was in fact connected with the outbreak, though not the cause of it in the gross manner which has been indicated."

Counsel's Concluding Statements

FOR THE CROYDON LOCAL MEDICAL AND PANEL COMMITTEE AND THE CROYDON DIVISION OF THE BRITISH MEDICAL ASSOCIATION

Mr. Forbes said that in the critical attitude of the Croydon doctors there was nothing in the nature of an attack on the medical officer of health. It was apparent to everybody that Dr. Holden was a loyal, able, and industrious servant of the corporation and in the witness-box a truthful and careful witness. But the question was bound to come up how far coöperation between medical officer and practitioners was sufficient and satisfactory and why there had been no more in the way of conference between them, and the practitioners thought it far better for their comments to reach the tribunal direct and, as he put it, chlorinated to their own standard. There had been in fact no consultation with any committee in the early days and local practitioners had wondered why. In its issue of Nov. 27th, 1937, the *Medical Officer* had said "We submit, rather we proclaim, that it is neither usual nor useful for the medical officer of health in times of urgency to confer with representatives of the local medical profession." Dr. Holden, asked if he subscribed to that, had replied that if a local committee had been available he would have been happy to have accepted and to have sought their help, subject to the consent of the public health committee. But he took what Croydon practitioners regarded as a narrow view. There being no such standing committee at the time of this outbreak he did not think there was any way in which he could help practitioners by meeting them in conference. Mr. Forbes submitted that Dr. Holden could not have found a better committee to coöperate with than the local medical and panel committee. They were now prepared to set up any standing committee which the corporation would recognise. According to the town clerk the corporation would now consider coöpting practitioners upon the public health committee. Referring to a resolution passed by the monthly meeting of the Croydon division of the B.M.A. Mr. Forbes said the local doctors did not now want to complain either that Dr. Holden did not send a letter to more than 26 doctors on Nov. 1st, or to more than 140 on Nov. 4th. He was in difficulties and was feeling his way.

FOR THE SANITARY INSPECTORS

Mr. Talbot submitted that if the theory was accepted that infection was communicated by a workman in

the well, and disseminated because the water was not then chlorinated, no responsibility could attach to the sanitary inspectors. There had been criticism of the camping site, but in the last resort the only possible way of getting rid of it was by proceedings for nuisance, and as far as they knew the ground was carried on in a perfectly proper manner. The only danger to health would have arisen not from anything done on the site but by reason of the fact that the prosecutors had a well some distance off. Cutting off the water was a matter of policy for the corporation to decide. The making of cesspools could not be prevented under existing law if no sewer was available. They could only be dealt with by sanitary inspectors if they overflowed or leaked, and there was no evidence of that kind except at the Addington Lodge farm where apparently the effluent from the cesspool had only penetrated a few feet into the ground. The latrine at the open-air school was under the jurisdiction of the education authority. Contamination of the water-supply being discovered, it had been suggested that the sanitary inspectors did not soon enough warn the inhabitants to boil their water, but, on the weight of evidence, infection took place during September and October and had ceased by Nov. 1st, on which day the water began to be chlorinated and in those circumstances it would have done no good to boil the water. Complaint had been made that warning was not given to empty storage tanks in unoccupied houses, but the typhoid bacillus had a very short life in water and nobody in fact had contracted the disease from that cause.

FOR THE CHIEF WATER ASSISTANT

Mr. Dingle Foot began by submitting that the evidence had not definitely excluded the reservoir as the source of infection. Mr. Brandram-Jones had been in the service of the corporation from September, 1935, until Oct. 16th, 1937. He succeeded Mr. Ord as chief water assistant but owing to Mr. Ord's fatal illness was not able to take over personally from him. Nothing in his training had made him familiar with chemistry or bacteriology. When employed by the Metropolitan Water Board he had no responsibilities for chlorination. The nature and extent of his duties were determined from time to time by the borough engineer. As regards taking of samples, he continued the system which he found in operation, the sampling being done by Mr. Ringham in the way prescribed by the Ministry of Health. His analyses were presented each month to the medical officer, the borough surveyor, and the water committee, and if instructions were not being carried out the surveyor had every opportunity of discovering the fact. No explicit instructions had been given him to increase the number of samples—at any rate he had no recollection of receiving any. How then did the misunderstanding arise as a result of which no sample was taken of the low water in Addington well from April till October, 1937? Actually Mr. Ringham had wrongly supposed that he was absolved from taking them from the well and took them from other wells thereafter. Mr. Brandram-Jones was not alone in omitting to notice what had happened. On the principal question, that of chlorination, Mr. Dingle Foot recalled that up till July, 1936, Addington had been chlorinated for one or two days each month and that other wells had not been chlorinated at all. Until then the official view was rather against chlorination; for the improvement after July, 1936, Mr. Brandram-Jones was largely responsible. In the summer of 1936 he was in touch with the M.W.B. examination department about the chlorination and discussed the correct dosage with Dr. Holden.

As regards chlorination before September, 1937, Mr. Dingle Foot summarised the position in this way: (1) In July, 1936, Mr. Boast and Dr. Holden agreed in Mr. Brandram-Jones's hearing that the normal dose should be 0·05 part per million. (2) Mr. Boast and Dr. Holden agreed that when there was any reason for special precautions this dose should be stepped up, and this was done on two occasions. (3) Mr. Brandram-Jones had been led to suppose that the practice of keeping the chlorination plant running at 0·05 part per million was not as a safeguard but simply in order that an effective dose could be given at once if desirable. Mr. Dingle Foot submitted that it had not been suggested by any witness that Mr. Brandram-Jones had ever been told that he must keep the chlorinator working continuously, so that he had not been guilty of any breach of specific instructions.

On Sept. 16th Mr. Brandram-Jones and Mr. Ellis had made the discovery after careful inspection of the lay-out of the pipes at Addington that it was not possible, with both engines working simultaneously, to pass any of the water through the filters. In their subsequent interview with Mr. Boast they supposed they had made the position clear to him and that he realised that working both engines would mean cutting out the filters. He did not suggest that cutting out the chlorinator was ever mentioned at that interview, and there was no doubt that Mr. Boast did not realise that this was involved in cutting out the filters. In view of the close connexion between them, however, it was a reasonable conclusion to draw that anyone who authorised those under him to do the one was also authorising them to do the other. Mr. Brandram-Jones's attitude to chlorination was guided by the analyses. In 1936 these had given cause for alarm, and in August he had stepped up the dose of chlorination; but in August, 1937, the position was entirely different and the reports on the samples from all the wells in that month had been uniformly satisfactory.

With reference to the use of a bucket by the workmen for urination while down the well, Mr. Dingle Foot suggested that it was possible that the men might not have appreciated that the instructions they received to come to the surface in order to use the lavatory referred to urination at all. Seeing that coming to the surface involved an extremely unpleasant journey in a barrel which had already caused the death of one man and took some 12 minutes, there would be a strong temptation to disobey instructions and use the bucket in the adit. In any case every possible care had been taken in using the bucket and in carrying it up in the skip. Dr. Conybeare did not think that the infection could be attributed to spilling urine in the well, so that if every workman had come to the surface for urination the result would have been the same. He hoped the tribunal could come to the conclusion that Mr. Brandram-Jones and Mr. Ellis had faithfully carried out the instructions which had been given to them by those in authority.

FOR THE BOROUGH ENGINEER

Mr. Thesiger, who appears for the borough engineer, said it must not be lost sight of that Mr. Boast was primarily the borough surveyor, and that his academic knowledge of water had to be combined with knowledge of many other subjects—architecture, street-planning, drainage, and the like. Medical knowledge

or the danger of the urination or excretion of a carrier were not likely to be present in his mind. Mr. Thesiger wondered whether it did not seem that the water undertakings of an authority should not be controlled by a specialist. It was a matter of more than engineering; some knowledge of bacteriology might well be desirable. Mr. Boast had taken over his duties in 1935 from Mr. Carter who had had experience of the wells of Croydon for 30 years, and he had not been warned to keep any special eye on the Addington gathering ground, and at that time chalk water-supplies, as Dr. Suckling had said, enjoyed a good reputation for purity. The farms on the gathering ground had been there for many years. As regards cesspools on the gathering ground, only six new houses had been built there since 1931 and in each case their cesspools had been properly supervised and made according to the by-laws of the corporation. Mr. Boast had carried on the system of monthly analyses which had been the practice for many years. He had inherited a system under which very little chlorination had been practised at all, and it was only because in 1936 the M.O.H., Mr. Boast, and Mr. Brandram-Jones had started the practice of chlorination and had introduced more modern instruments at the pumping stations that the omission of chlorination at any time had become noticeable. In November, 1936, Mr. Boast had said, in reply to a memorandum from Dr. Holden: "I am arranging in future, in addition to test analyses of the raw water, for the actual water put into the mains to be analysed after chlorination." In introducing this change, Mr. Boast was not meaning to drop the testing of the raw water, but was introducing an improvement on the pre-existing practice. If the testing of the raw water had been continued once a month at each well, there was no evidence that anything would have come to light with regard to infection by a carrier in October, 1937, in time for anything to have been done.

In the matter of the instructions about the work which was to be done in the well there was a difference in the recollection of Mr. Boast and Mr. Brandram-Jones, but there was no doubt that the instructions to pump to waste while the men were in contact with the water, to pick men who appeared to be physically fit, and to wear special clothing had been carried out.

Coming to the final matter on which he anticipated that some criticism might be made of Mr. Boast, the question of the cessation of chlorination, it was quite clear that on Oct. 31st Mr. Boast, who had been left without a water assistant since Oct. 16th, was under the impression that chlorination was proceeding at Addington, and the purpose of his telephone conversation with Mr. Brandram-Jones had been to find out the dosages being used for the high- and low-level supplies. He had told Dr. Holden with surprise that evening that in fact there was no chlorination at Addington, and, although at the time this did not seem to be a vital matter, it had been attended to with promptness and the very next day arrangements had been made to put in a new chlorinator to catch the water as it went into supply.

Mr. Murphy asked how it was that, when Mr. Boast knew that chlorination had stopped at Addington, a bacteriological examination of the Addington well water had not been done until Dr. Conybeare appeared on the scene on Nov. 3rd. Mr. Thesiger replied that at that time it had not occurred to Mr. Boast to connect up the absence of chlorination with the typhoid cases, and a sample had been taken on Oct. 29th, on which a report was being awaited; he would not have thought that the taking of another sample could produce any quicker result. The importance of the fact that men had been working in the Addington well had never occurred to Mr. Boast.

"We all know now," said Mr. Thesiger, "that one touch from the soiled hand of a man on the brickwork to which the water was rising may have caused this infection, and I ask you to say that that, clearly, on the evidence, was never present in Mr. Boast's mind."

Mr. Murphy remarked that the true answer to his question might well be that water was not suspected until Dr. Conybeare arrived.

FOR THE MEDICAL OFFICER OF HEALTH

Mr. Sandlands said he was not concerned with the question whether there should be a statutory obligation on all local authorities to start medical committees. Dr. Holden had said that if a committee had been in existence he would gladly have coöperated with it, but he did not see how he could call one together in the middle of an outbreak. He proposed to deal with the possible causes of the outbreak and to inquire whether Dr. Holden could be blamed for any of them; then to ask whether he had taken all the steps he ought to localise the trouble quickly. A medical officer of health had various statutory duties but these did not include looking after the water department. According to the regulations of 1935: "He must inform himself as far as practicable as to all matters affecting or likely to affect public health in the district, and be prepared to advise the local authority on those matters." The phrase "as far as practicable" must mean: "Put yourself in the circumstances of the medical officer of health in that particular place, see what his predecessor has done, see what he has been asked to do, and see what in fact he has done." When in 1933 the Ministry of Health had inquired into the duties of the M.O.H. in Croydon they had never suggested that he was under any obligation to carry out any water duties. Dr. Holden had said that his duty was to keep his eyes open and inform himself generally of the position. From time to time he had inspected the gathering grounds and had made recommendations to the borough engineer. In Croydon, at any rate, the engineer was the senior officer.

The most likely cause of the outbreak, Mr. Sandlands submitted, was infection of the water in the Addington well by a carrier. How was Dr. Holden to be held responsible for that? He did not know that work was being done in the well; he did not know under what conditions it was going on, and no one had thought of asking him whether they should work without chlorination in September and October. It was an unhappy chapter of accidents for which no one could be blamed. Nobody until this case appeared to have heard of having a medical test of men working in a well. Dr. Suckling had said that it would be a good thing in the future to ask men if they had had typhoid, but he had not heard of any instance where it had been done in the past.

Passing to the gathering grounds, Mr. Sandlands said that the camps either inside or outside the borough had never been brought to Dr. Holden's notice. It was unlikely that anything would get to the well from the manure heap at Fisher's farm, and if it did it would be harmless because it was from animal and not human excreta. Dr. Holden had said if there had been a case of typhoid at this farm it would have been a point of danger which he would have watched very closely. If there had been such a case it would have been taken to hospital and all

kinds of precautions would have been taken, and no doubt a heavy dose of chlorination would have been put into the well. Until October, 1937, there had been no power for the local authority to connect the drains from the farm to a sewer. Now, under the Act of 1936, there was such power and, as a precaution, it had been done.

With regard to cesspits, Dr. Holden had said that the sanitary inspectors kept an eagle eye on them, and there had been no complaints. Referring to the latrine at St. Giles' School, which Dr. Suckling had regarded as the most likely potential source of infection from the gathering ground, Dr. Holden had said: "If I had known that the thing was there it would not have been allowed to remain." Mr. Murphy interposed to say that this latrine was certainly a potential menace, being almost directly over an adit. Even the borough engineer did not know it was there, Mr. Sandlands remarked. The school belonged to the education department and they had entrusted the work to a contractor. With regard to any of these potential sources of infection, he submitted, Dr. Holden could not be held to blame, and, even if the tribunal found that all of them had contributed, the governing answer was that Dr. Holden believed that chlorination was in progress.

The analyses from August, 1936, until Oct. 29th, 1937, had not shown that the water required any treatment other than it was then getting. Thereafter Dr. Holden, far from doing anything wrong, had done the best thing possible. The question of flies had had to be investigated, laundries communicated with, and Dr. Holden had even, at the request of local doctors, visited fifty patients in their homes. This showed the great thoroughness of his work and the cordial coöperation there was between him and the local doctors.

FOR THE SOUTH CROYDON TYPHOID OUTBREAK COMMITTEE

Mr. Lyons said that in Croydon there was a water committee and a public health committee which dealt with specialised subjects, but no chairman or member of these committees had thought fit to come to the inquiry and volunteer information. "Is it the duty of any responsible person," Mr. Lyons asked, "either as chairman or vice-chairman or member of a public body to say that he or she will accept whatever honour and authority that office may give, but yet will not accept any responsibility for the paid officers of the department or for any errors, inaccuracies, or neglect?" The chairman of these committees, said Mr. Lyons, had paid no attention whatever to what was being done by their employees. It had been urged on behalf of the borough engineer that he had many offices and a lot of work to do. What were the local authority doing in appointing a man who knew nothing about the real work in the direction of water-supplies? Was it fair for Dr. Holden and Mr. Boast to be summoned to the inquiry, and for the local authority who appointed them to run away and say that it was no business of theirs? There was, Mr. Lyons maintained, clear evidence of absolute non-coöperation between these two officers—not the slightest attempt to get together to realise the dividing lines of their respective offices and where those lines converged. Between two stools no one seemed to have cared where the public interest fell.

FOR THE CROYDON CORPORATION

Sir Walter Monckton said the local authority had put before the tribunal all the information at its disposal. He took entire responsibility for the fact that certain persons to whom Mr. Lyons had referred had not gone into the box. The public health and water committees had accepted the recommendations of the responsible officers, but they had no wish to shelter behind them. There was obviously a moral responsibility on the committees and on the local authority which he hoped no word of his had sought to avoid.

Sir Walter replied to two questions put by Mr. Murphy. (1) Were the corporation slow in not tackling the whole water-supply problem between Oct. 29th and Nov. 3rd? (2) If slowness were attributable had it any effect on the outbreak and its extent? He recalled Sir William Willcox's view that the cause of the epidemic was discovered very quickly and that from a scientific point of view the diagnosis was quickly made. If it was true, as he believed, that infection had reached each person by Nov. 1st, it was more material to look at the period Oct. 30th to Nov. 1st and then it could hardly be contended that Dr. Holden was slow in looking at the water-supply. Even should the tribunal attribute to him some slowness there was nothing to show that any case of typhoid resulted from it. In the matter of chlorination Sir Walter reminded the tribunal that it was first introduced in the borough in 1936 when there had been some adverse analyses; it was not regarded as something permanent but as something to be used for a specific purpose when the analyses were bad.

Close of the Inquiry

When the inquiry ended on Wednesday morning, after sitting for 17 days, the chairman, Mr. Murphy, said that a report would be presented with the least possible delay.

INFECTIOUS DISEASE

IN ENGLAND AND WALES DURING THE WEEK ENDED JAN. 1ST, 1938

Notifications.—The following cases of infectious disease were notified during the week: Small-pox, 0; scarlet fever, 2383; diphtheria, 1417; enteric fever, 34 (23 "expected"); pneumonia (primary or influenzal), 1648; puerperal pyrexia, 163; cerebro-spinal fever, 35; acute poliomyelitis, 6; encephalitis lethargica, 1; dysentery, 247; ophthalmia neonatorum, 104. No case of cholera, plague, or typhus fever was notified during the week.

It should be recalled that all these figures are artificially enhanced by the incompleteness of returns in the previous (holiday) week.

The number of cases in the Infectious Hospitals of the London County Council on Jan. 7th was 4205, which included: Scarlet fever, 806; diphtheria, 1474 (diphtheria carriers, 30); measles, 496; whooping-cough, 345; puerperal fever, 14 mothers (plus 12 babies); encephalitis lethargica, 283; poliomyelitis, 3. At St. Margaret's Hospital there were 20 babies (plus 10 mothers) with ophthalmia neonatorum.

Deaths.—In 125 great towns, including London, there was no death from small-pox, 7 (1) from enteric fever, 45 (7) from measles, 7 (0) from scarlet fever, 19 (3) from whooping-cough, 43 (8) from diphtheria, 72 (17) from diarrhœa and enteritis under two years, and 59 (10) from influenza. The figures in parentheses are those for London itself.

Six deaths from typhoid were reported from Croydon. Liverpool had 19 deaths from measles, Manchester 7, no other great town more than 2. Liverpool also reported 4 deaths from whooping-cough. Diphtheria was fatal in 26 great towns, Liverpool reporting 7 deaths; no other great town more than 2.

The number of stillbirths notified during the week was 237 (corresponding to a rate of 39 per 1000 total births), including 34 in London.

PUBLIC HEALTH

Prevalence of Dysentery

THERE is again during 52nd week a reduction in the number of cases of dysentery notified. The return for 1937 is now complete and the notifications of dysentery in England and Wales during the 52 weeks of 1937 add up to 4025. Assuming, as seems likely, that the number of institutional cases has not changed greatly, and deducting (say) 400 from the total, in order to arrive at the figure for the extra-hospital population, we find (as was suggested on p. 1462 of an earlier issue) that the general prevalence throughout the country in 1937 was at least tenfold what it was in 1934, probably near twelvefold. In the week under review it is still the home counties which account for the majority of the reported cases, the figures being: London 77, Middlesex 22, Kent 13, Essex 12, Sussex West 9, Surrey 8; other counties still with unusual numbers to report being Devon 5 (scattered), Lancaster 10, Norfolk 19 (Norwich 17), Nottingham 13 (county borough), Oxford 6 (county borough), Somerset 8 (scattered), Stafford 10 (scattered), Wilts 6 (Swindon 5), Glamorgan 9 (Cardiff 7, Swansea 2).

NOTIFIABLE DISEASES DURING 1937

The Registrar-General's returns now being complete for the 52 weeks of 1937, we give the total numbers of certain infectious diseases notified in the 125 great towns (including London), and the proportion which proved fatal, with comparative figures for 1936.

Disease.	Cases.		Deaths.	
	1936.	1937.	1936.	1937.
Enteric fever ..	1,414	1,283	138	110
Small-pox	12	0	0	0
Scarlet fever ..	55,999	53,927	225	179
Diphtheria	34,529	38,266	1,705	1,603
Measles	—	—	1,896	617
Influenza	—	—	2,780	8,325

Measles and influenza not being notifiable the number of cases is not known.

OBITUARY

THOMAS TENDRON JEANS, C.M.G., M.B. Lond.

Surgeon Rear-Admiral Jeans, who died at Southsea on Jan. 4th in his sixty-seventh year, had spent 32 years of his life in the Navy, and he has left all that needs to be told of his life at sea in his "Reminiscences of a Naval Surgeon" published in the year following his retirement. Jeans qualified M.B. Lond. in 1893, entered the Navy in the following year, saw active service at Manila during the Spanish-American war, and as surgeon of *Monarch* was present with the Naval Brigade at Paardeberg and Bloemfontein; he gives a vivid picture of Cronje's laager after its surrender. Later he served in the flag-ship of the West Indian station, visiting Venezuela among other places; and when war broke out in 1914 he was on the East Indian station and went with the flag-ship to cover the landing at Gallipoli. Jeans retained to the end of his life a staunch love of the Navy, although he was critical of a system which he thought prevented the junior executive officer from exercising independent thought and sharpening his wits against those of others. Earlier in his life he had written a series of stories for boys about the Navy, including "Ford of H.M.S. Vigilant," "Mr. Midshipman Glover, R.N.," and "John Graham, Sub-Lieutenant, R.N." The last appeared in 1913 and he did not resume his service yarns when the war was over, as he felt that the prospects in the Navy were not then what he would have wished them to be. From 1919 to 1922 he was in charge of the Naval Hospital at the Cape and for some years before his retirement he was at the Plymouth Hospital. Jeans was made C.M.G. in 1919 for valuable services in various theatres of the war.

WILLIAM L. M. GABRIEL, M.B. Leeds, B.Hy. Durh.

Dr. Gabriel, who died on Dec. 30th, was 46 years of age and had been an assistant medical officer for Northumberland since 1920. He was a Yorkshireman, born at Keighley, and was a student at Leeds University at the outbreak of the late war. In August, 1914, he accepted a commission in the West Yorkshire Regiment as a combatant officer and went to France with the 2nd battalion early in 1915. A few months later he was badly wounded at Aubers ridge and during convalescence in England he was able to complete his medical studies at Leeds, graduating with second-class honours in 1917. After a short time as resident medical officer at the Leeds Public Dispensary, he took a commission in the Royal Army Medical Corps (Special Reserve) and became medical officer in charge of the Royal Army Ordnance Department at Chilwell. Joining the public health service of Northumberland after the war he acted as medical officer of health for the Gosforth urban district council from 1932 onwards, but at the time of his death had just resigned this appointment in order to take over the duties of senior assistant county medical officer. A colleague writes of the bright future that appeared to be in front of him, of his charm, his loyalty, and the devotion he gave to his work. In leisure hours this sense of service made him a keen territorial, and he succeeded to the command of the 149th Field Ambulance Brigade two years ago, later representing it at the Coronation and receiving the Territorial Decoration.

Dr. Gabriel leaves a widow with one young son.

GERMAN JOURNAL FOR RHEUMATISM.—Under the title of *Zeitschrift für Rheumaforschung* a journal will appear this year edited by Dr. P. Köhler (Bad Elster) and Prof. R. Jürgens and Dr. H. Kaether (Berlin), which will seek to collect and epitomise the many papers on rheumatism which are being published in Germany and elsewhere. The publisher is Theodor Steinkopff of Dresden.

NATIONAL INSTITUTE OF ECONOMICS AND SOCIAL RESEARCH.—Under this name an institute has been formed which will investigate problems of contemporary human society. Grants for the purpose have been made by the Sir Halley Stewart trust, the trustees of the late Lord Leverhulme, the Pilgrim trust, and the Rockefeller Foundation. Sir Josiah Stamp is to act as president, and Prof. N. F. Hall, who is head of the department of political economy in the University of London at University College, has been invited to become its first director. The offices of the institute are at 32, Gordon-square, London, W.C.1.

MEDICAL NEWS

University of London

At recent examinations the following candidates were successful :—

M.D.

Branch I (*Medicine*).—John Apley, Univ. Coll. Hosp.; C. G. Baker, Guy's Hosp.; Jean M. Cass and Lynette Dowsett, Roy. Free Hosp.; E. A. Hardy, Westminster Hosp.; G. W. Hearn, St. Thomas's Hosp.; C. A. Lillicrap and W. N. Mann, Guy's Hosp.; S. K. Squires, St. Mary's Hosp.; and R. J. Vakil, St. Thomas's Hosp.

Branch II (*Pathology*).—D. G. ff. Edward, St. Bart.'s Hosp.

Branch IV (*Midwifery and Diseases of Women*).—T. L. S. Baynes, St. Bart.'s Hosp.; P. I. Boyd (University medal), Univ. Coll. Hosp.; and Anne A. Craig, Roy. Free Hosp.

Branch V (*Hygiene*).—Moses Sendak (University medal), St. Mary's Hosp.

The following have been recognised as university teachers at the schools indicated: Dr. P. M. F. Bishop, Dr. E. R. Boland, Mr. Kenneth Hooper, Dr. A. D. Marston, Mr. H. L. Messenger, Dr. C. Keith Simpson, and Mr. T. T. Stamm (Guy's Hospital); Mr. P. B. Ascroft (Middlesex Hospital); Dr. Thomas Tennent and Mr. W. N. L. Wade (King's College Hospital).

Applications are invited for the Geoffrey E. Duveen travelling studentship, of the value of £450, for research in any aspect of otorhinolaryngology.

The studentship is tenable in the first instance for one year, part of which must be spent abroad, but its tenure may be extended to allow the student to undertake research at the Royal Ear Hospital or elsewhere. Further particulars may be had from the academic registrar, University of London, W.C.1, to whom applications should be sent before Dec. 31st, 1938.

The annual conversazione of the university will be held on Thursday evening, March 17th. Tickets may be obtained from the clerk of convention at the University, W.C.1.

At 5 P.M. on Mondays, beginning on Jan. 17th, at University College, Gower-street, W.C., Mr. H. R. Ing, D.Phil., will give six lectures on chemical structure and pharmacological action.

Royal Institute of Public Health and Hygiene

On Wednesday, Jan. 19th, at 3.30 P.M., Sir Walter Langdon-Brown will give the first of a series of lectures on national health. He will speak on diet and the national health, and subsequent lectures will be announced week by week in our Medical Diary. All the lectures will be held in the lecture hall of this institute, 28, Portland-place, London, W.

B.M.A. Scholarships and Grants in Aid of Research

The council of the British Medical Association is prepared to receive applications for research scholarships and grants for assistance of research in connexion with disease.

Scholarships.—An Ernest Hart memorial scholarship (£200 per annum), a Walter Dixon scholarship (£200 per annum), and three research scholarships (each £150 per annum). These scholarships are given to candidates whom the Science Committee of the Association recommends as qualified to undertake research in any subject (including State medicine) relating to the causation, prevention, or treatment of disease. Preference will be given, other things being equal, to members of the medical profession. Each scholarship is tenable for one year, commencing on Oct. 1st, 1938. A scholar may be reappointed for not more than two additional terms, and is not necessarily required to devote the whole of his or her time to the work of research, but may hold a junior appointment at a university medical school, or hospital, provided the duties of such appointment do not interfere with the work as a scholar.

Grants.—Grants for the assistance of research in the causation, treatment, or prevention of disease. Preference will be given, other things being equal, to members of the medical profession and to applicants who propose as subjects of investigation problems directly related to practical medicine.

Application for scholarships and grants must be made not later than May 7th, on the prescribed form, a copy of which will be supplied on application to the secretary of the Association, B.M.A. House, Tavistock-square, London, W.C.1. Applicants are required to furnish the names of three referees who are competent to speak as to their capacity for the research contemplated.

Society for Constructive Birth Control

At a meeting of this society on Wednesday, Jan. 19th, at 8 P.M., at Manson House, 26, Portland-place, London, W., Dr. John Hannan will speak on the dangers of abortion.

Institute of British Surgical Technicians

On Friday, Jan. 21st, at 8.15 P.M., at the Welbeck Hotel, Welbeck-street, London, W., Mr. A. E. Mortimer Woolf will give a lecture on general abdominal technique. Tickets may be had from the secretary of the institute, 6, Holborn-viaduct, London, E.C.1.

Swedish Woman Professor of Medicine

Dr. Nanna Svartz-Malmberg has been appointed professor of medicine at the Stockholm Caroline Institute by the Swedish Government. This is the first time in Sweden that a woman has been appointed professor. Dr. Svartz-Malmberg is known especially for her work on the intestinal bacteria and their influence on rheumatic diseases.

The Film in Biology

Three lectures on this subject will be given on Thursdays, Jan. 27th, Feb. 3rd and 10th, at 5.15 P.M., at the Royal Institution, 21, Albemarle-street, London, W. Mr. T. H. Gillespie will speak on the king penguin, Mr. E. J. Salisbury, F.R.S., on plant life, and Mr. H. R. Hewer on embryology. Each lecture will be illustrated.

State Veterinary Service

The following appointments have been made by the Ministry of Agriculture and Fisheries in the new State veterinary service: Sir John Kelland, M.R.C.V.S., chief veterinary officer; Mr. D. A. E. Cabot, M.R.C.V.S., and Mr. E. C. Lloyd, M.R.C.V.S., deputy chief veterinary officers; and Captain W. G. Wragg, M.R.C.V.S., chief superintending inspector.

British Postgraduate Medical School

On Feb. 2nd, 9th, and 16th, at 4.30 P.M., Dr. Dorothy Russell will lecture at the school on the pathology of diseases of the nervous system, and on Feb. 4th, 11th, and 18th, at 2.30 P.M., Prof. Lambert Rogers will speak on surgery of the spinal cord and peripheral nerves. Four lectures on the hygiene of the new-born child will be given at 3.30 P.M. on Feb. 3rd, 10th, 17th, and 24th, by Prof. V. H. Mottram and Dr. Alan Moncrieff. Tickets may be had from the dean of the school, Ducane-road, London, W.12.

Society for Relief of Widows and Orphans of Medical Men

At a quarterly court held on Jan. 5th the president, Mr. V. Warren Low, being in the chair, two new members were elected and the death of one reported. A sum of £2183 15*s.* was voted for the payment of the half-yearly grants to the sixty-one widows and five orphans in receipt of relief. Three special grants each of 50 guineas per annum were made to three orphans, who had reached the age of 16 when ordinary grants cease, to enable them to continue their education. A first application for relief was received from the widow of a deceased member and she was voted £50 per annum from the ordinary funds and £25 per annum from the Brickwell Fund. Receipt of a further legacy (of £168) was reported from the executors of the late George Cattlin, bringing the total amount received from this source up to £4243. It was decided to celebrate the 150th anniversary of the foundation of the society by a dinner to be held in London on Saturday, Oct. 29th; the society was founded at a meeting held at the Gray's Inn Coffee-house on Oct. 29th, 1788, over which Sir George Baker, Bt., F.R.S., physician to King George III, presided. Membership is open to any registered medical man, who at the time of his election is residing within a 20-mile radius of Charing Cross. Particulars from the secretary at 11, Chandos-street, London, W.1.

Medical Diary

January 17th to January 22nd

SOCIETIES

ROYAL SOCIETY OF MEDICINE, 1, Wimpole-street, W.
TUESDAY.
5.30 P.M., general meeting of fellows.
Pathology. 8.15 for 8.30 P.M.
Laboratory meeting at St. Mary's Hospital, W.
THURSDAY.
Dermatology. 5 P.M. (Cases at 4 P.M.)
Dr. H. MacCormac: 1. Chauffard-Still-Felty Syndrome. Dr. L. Forman: 2. ? Scleroderma. Dr. H. C. Semon: 3. Lichen Planus of the Tongue, Finger, and Toe-nails. Dr. G. B. Dowling: 4. Premycotic Erythema. 5. Acrodermatitis Pustulosa Hæmalis.
Neurology. 8.30 P.M.
Dr. H. G. Garland: Rôle of Trauma in the Pathology of Organic Disease (excluding Epilepsy).
FRIDAY.
Physical Medicine and Dermatology. 4.30 P.M.
Dr. H. W. Barber, Dr. Elizabeth Hunt, Dr. Kerr Pringle, and Dr. W. Yeoman: Skin Manifestations in Rheumatism.
Radiology and Obstetrics and Gynæcology. 8.30 P.M.
Dr. W. M. Levitt, Dr. J. F. Bromley, and Mr. Leonard Phillips: Radiotherapy in Non-malignant Gynæcological Disorders. Mr. Malcolm Donaldson, Miss Louisa Martindale, and Mr. L. Carnac Rivett will also speak.

HUNTERIAN SOCIETY.
MONDAY.—9 P.M. (Mansion House, E.C.), Dr. Gustav Singer: Functional Disorders of the Intestine. (Hunterian lecture.)

CHELSEA CLINICAL SOCIETY.
TUESDAY.—8.30 P.M. (Rembrandt Hotel, Thurloe-place, S.W.), Dr. James Davidson: The Work of a Forensic Science Laboratory.

NORTH LONDON MEDICAL AND CHIRURGICAL SOCIETY, Royal Northern Hospital, N.
THURSDAY.—9.15 P.M., Sir Walter Langdon-Brown: The Dead Hand in Medical Science.

SOCIETY OF RADIOTHERAPISTS, 11, Chandos-street, W.
FRIDAY.—5.30 P.M., Dr. N. S. Finzi and Mr. J. E. Roberts: Impressions of the Chicago Congress and of Radiotherapy in America.

SOCIETY OF MEDICAL OFFICERS OF HEALTH, 1, Thornhaugh-street, W.C.
Maternity and Child Welfare Group.
FRIDAY.—8.30 P.M., Dr. Stella Churchill: Eugenics and Maternity and Child Welfare Work.

EUGENICS SOCIETY.
TUESDAY.—5.15 P.M. (rooms of the Royal Society, Burlington House, Piccadilly, W.), Dr. G. F. McCleary: Population Problems in the British Commonwealth.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE, Manson House, 26, Portland-place, W.
THURSDAY.—7.45 P.M., Demonstration. 8.15 P.M., Prof. R. Brunel Hawes (Singapore): Acute Cardiac Beri-beri. Prof. R. A. Peters, F.R.S.: The Biochemistry and Physiology of B_1 Deficiency. Dr. B. S. Platt (Shanghai): Pyruvic Acid in Beri-beri.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland-place, W.
WEDNESDAY.—3.30 P.M., Sir Walter Langdon-Brown: Diet and the National Health.

BRITISH INSTITUTE OF RADIOLOGY, 32, Welbeck-street, W.
THURSDAY.—8.30 P.M., Dr. J. F. Brailsford: Paget's Disease of Bone, its Frequency, Diagnosis, and Complications.

LECTURES, ADDRESSES, DEMONSTRATIONS, &c.

ROYAL COLLEGE OF SURGEONS OF ENGLAND, Lincoln's Inn-fields, W.C.
MONDAY.—5 P.M., Mr. J. Cole Marshall: Surgical Treatment of Detachment of the Retina.
WEDNESDAY.—5 P.M., Mr. R. T. Payne: Cancer of the Stomach as a Surgical Problem.
FRIDAY.—5 P.M., Mr. R. C. Brock: The Pathology, Diagnosis, and Treatment of Intrapulmonary New Growths. (Hunterian lectures.)

UNIVERSITY COLLEGE, Gower-street, W.C.
MONDAY.—5 P.M., Mr. H. R. Ing, D.Phil.: Chemical Structure and Pharmacological Action. (First of six lectures.)

BRITISH POSTGRADUATE MEDICAL SCHOOL, Ducane-road, W.
TUESDAY.—4.30 P.M., Prof. Arthur Ellis: Nephritis.
WEDNESDAY.—Noon, clinical and pathological conference (medical). 2 P.M., Mr. E. J. King, Ph.D.: Tests of Liver Function. 3 P.M., clinical and pathological conference (surgical). 4.30 P.M., Dr. Cuthbert Dukes: The Pathology of Calculous Disease of the Urinary Tract.
THURSDAY.—2.15 P.M., Dr. Duncan White: radiological demonstration. 3.30 P.M., Mr. J. Bright Banister: Cæsarean Section.
FRIDAY.—2 P.M., clinical and pathological conference (obstetrics and gynæcology). 2.30 P.M., Mr. C. Naunton Morgan: Diseases of the Rectum.
Daily, 10 A.M. to 4 P.M., medical clinics, surgical clinics and operations, obstetrical and gynæcological clinics and operations.

WEST LONDON HOSPITAL POST-GRADUATE COLLEGE, Hammersmith, W.
MONDAY.—10 A.M., Dr. Post: X ray film demonstration, skin clinic. 11 A.M., surgical wards. 2 P.M., operations, surgical and gynæcological wards, medical, surgical, and gynæcological clinics. 4.15 P.M., Mr. Green-Armytage: Trespass.
TUESDAY.—11 A.M., surgical wards. 2 P.M., operations, medical, surgical, and throat clinics. 4.15 P.M., Dr. Coden: Choice of Anæsthesia.
WEDNESDAY.—10 A.M., children's ward and clinic. 11 A.M., medical wards. 2 P.M., gynæcological operations, medical, surgical, and eye clinics. 4.15 P.M., Mr. Harvey Jackson: Neuro-surgical Investigations.
THURSDAY.—10 A.M., neurological and gynæcological clinics. Noon, fracture clinic. 2 P.M., operations, medical, surgical, genito-urinary, and eye clinics.
FRIDAY.—10 A.M., medical wards, skin clinic. Noon, lecture on treatment. 2 P.M., operations, medical, surgical, and throat clinics.
SATURDAY.—10 A.M., children's and surgical clinics. 11 A.M., medical wards.
The lectures at 4.15 P.M. are open to all medical practitioners without fee.

FELLOWSHIP OF MEDICINE AND POST-GRADUATE MEDICAL ASSOCIATION, Wimpole-street, W.
MONDAY to SATURDAY.—ST. PETER'S HOSPITAL, Henrietta-street, W.C., all-day course in urology.—NATIONAL HOSPITAL FOR DISEASES OF THE HEART, Westmoreland-street, W., all-day course in cardiology.—ST. JOHN'S HOSPITAL, 5, Lisle-street, W.C., afternoon course in dermatology. (All open to non-members.)

LONDON SCHOOL OF DERMATOLOGY, 5, Lisle-street, W.C.
TUESDAY.—5 P.M., Dr. W. Griffith: Eczema.
WEDNESDAY.—5 P.M., Dr. I. Muende: Histopathology of Some Common Skin Diseases. (Chesterfield lectures.)

ST. GEORGE'S HOSPITAL, S.W.
THURSDAY.—5 P.M., Dr. Anthony Feiling: Neurological Demonstration.

HOSPITAL FOR SICK CHILDREN, Great Ormond-street, W.C.
THURSDAY.—2 P.M., Dr. B. E. Schlesinger: Infant Feeding on the Breast. 3 P.M., Mr. Charles Donald: Hydrocephalus and Spina Bifida.
Out-patient clinics daily at 10 A.M. and ward visits at 2 P.M.

ST. JOHN CLINIC AND INSTITUTE OF PHYSICAL MEDICINE, Ranelagh-road, S.W.
FRIDAY.—4.30 P.M., Dr. C. Worster-Drought: Physical Medicine in Diseases of the Nervous System.

CENTRAL LONDON THROAT, NOSE, AND EAR HOSPITAL, Gray's Inn-road, W.C.
FRIDAY.—4 P.M., Mr. W. A. Mill: Difficulty in Swallowing.

GLASGOW POST-GRADUATE MEDICAL ASSOCIATION.
WEDNESDAY.—4.15 P.M. (Ear, Nose, and Throat Hospital), Dr. Charles Macartney: Nasal Diseases.

Births, Marriages, and Deaths

BIRTHS

BEILBY.—On Jan. 4th, at St. Bartholomew's Hospital, the wife of Dr. F. J. Beilby—a son.
GERRARD.—On Jan. 6th, at Banstead, the wife of Dr. W. W. Gerrard—a son.
GORDON.—On Jan. 8th, at Sutherland-avenue, Maida Vale, the wife of Dr. R. M. Gordon, of Silver End, near Witham, Essex—a daughter.
MILL.—On Jan. 5th, at Welbeck-street, the wife of Mr. W. A. Mill, F.R.C.S.—a daughter.
SAVILLE.—On Jan. 6th, at Whitstable, the wife of Dr. J. E. Saville—a daughter.

MARRIAGES

WALTON—DOWNTON.—On Jan. 6th, at St. Botolph's Church, Morley, Wymondham, Capt. Henry Courtenay Mansel Walton, R.A.M.C., to Mary Geraldine, eldest daughter of the Rev. B. M. Downton, of Wymondham.

DEATHS

BERNAYS.—On Jan. 8th, at the Rectory, Finchley, Adolphus Vaughan Bernays, M.B. Camb., of Solihull, Warwickshire, son of the late Rev. L. J. Bernays, Rector of Stanmore.
BIDWELL.—On Jan. 10th, at Teignmouth, Lawrence Bidwell, M.R.C.S. Eng., Surg.-Comd., R.N. (retd.).
BLUCKE.—On Jan. 11th, at Denmark Hill, H. F. S. Blucke, M.R.C.S. Eng., late M.O.H., Borough of Shaftesbury.
HELM.—On Jan. 4th, at Carlisle, Robert Dundas Helm, M.D. Edin., J.P.
JEANS.—On Jan. 4th, at Southsea, Surg. Rear-Admiral T. T. Jeans, C.M.G., M.B. Lond.
THORNICRAFT.—On Jan. 7th, at Artillery Mansions, Thomas Charles Thornicraft, M.R.C.S. Eng. (late of Kobe, Japan), aged 92.
WILLIAMS.—On Jan. 8th, at Newport, Mon., Herbert Egerton Williams, M.R.C.S. Eng., in his 80th year.

N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages, and Deaths.

NOTES, COMMENTS, AND ABSTRACTS

DOCTOR AND PATIENT *

By Sir Maurice Cassidy, K.C.V.O., C.B., M.D., F.R.C.P.

SENIOR PHYSICIAN, ST. THOMAS'S HOSPITAL

From time to time we doctors, like most other men, let off steam by having a good grumble. For instance, we grumble about the vagaries of our patients, just as our patients grumble about the incompetence of their doctors. We grumble when the weather is fine and our patients are so healthy that we see starvation staring us in the face; and when the weather is bad we grumble again because we are run off our feet—and because the weather is so inclement. It is, in fact, seldom difficult to find something to grumble about, and it must be confessed that most of us derive a certain savage satisfaction from our grumblings. But there is one thing about which we never grumble—namely, the fact that we happen to be doctors; and this is a very agreeable reflection. We constantly encounter patients who tell us that they are bored by their work, or even loathe it; and we are very sorry for such unfortunates, for we doctors are to a man happy in our work, and I venture to say that most of us, if given the chance to start life afresh, would unhesitatingly choose to be doctors again, though possibly some of us would, with the experience we have gained, plan our professional lives a little differently.

The explanation of the doctor's contentment with his vocation is of course a very simple one. The man who does not like doctoring finds it out at an early stage of his career as a student, or his examiners find it out for him, and he switches off into the timber trade or some other more congenial occupation—possibly he writes a best seller about dishonest doctors! We are happy in our work for various reasons. Paramount, of course, must be the extraordinary satisfaction of helping others; of saving lives from time to time, and constantly of relieving suffering, physical or mental. Then how fortunate we are to continue to be students throughout our professional lives! Never a week passes but we learn something new and stimulating about the science or art of medicine, and to some few of us it may be granted to add a stone, if it be only a pebble, to the constantly expanding edifice which our profession has been engaged in building since the time of Hippocrates.

Finally, we have the inestimable advantage of intimate contact with all sorts and conditions of men. I doubt if any other profession offers such priceless opportunities of making firm and valued friendships. In this respect, as in many others, the general practitioner is more fortunate than the consultant, who sees, or should see, most of his patients once only, and very few of them more than two or three times a year. When the consultant accompanies a middle-aged or elderly practitioner on his round, I can assure you that he is often filled with envy as he sees how his host is greeted with obvious affectionate regard by all his patients. Many of them he has helped to bring into the world; he has seen them through the troubles of childhood and adolescence, and in due course he has perhaps assisted at the birth of *their* children. He has shared their happiness, and sometimes their sorrow, and they come to him for advice which is not always purely medical. To be able to achieve this happy relationship requires something more than professional erudition, and it is sometimes a little difficult to understand how it is that one doctor succeeds and another fails to win the confidence and affection of his patients. I venture to suggest that "a good bedside manner" is not the solution of the problem, and I am glad to think that the breezy type of practitioner, with his "Well, Mrs. Smith, and how are we this morning?" is rapidly becoming extinct. It seems to me that after professional competence which, of course, must always come first, sympathy and a sense of humour are the most essential qualities in a doctor. And by sympathy I do not mean maudlin sentimentality, but rather the capacity to put yourself in the patient's place and attempt to realise his attitude towards his illness, remembering always that, bluff though he may, anxiety, and often fear, haunt him.

THE DOCTOR AS PATIENT

When I first put up my brass plate, an old general practitioner said to me, "You will never succeed in practice unless you find that the majority of your patients leave your consulting-room looking ten years younger than they did when they entered it!" This very satisfactory phenomenon is one which is, I am sure, familiar to most of you. Another bit of advice which he gave me is not always easy to follow. It was to have, oneself, at least one acute and one chronic illness; and if one could undergo an abdominal operation, so much the better. Finally, he added, one should pass a catheter and an œsophageal tube on one's own vile body. I must confess, however, that I never screwed myself up to follow this advice in full, though I learnt much from the acute and the chronic illness which in the course of time fell to my lot. In passing, it is curious that we doctors make such thoroughly bad patients when we fall ill ourselves, for it is notorious that we do so. It is, I suppose, very difficult for us to dissociate ourselves from the technical aspect of our malady. We score off the layman in so far as not to be anxious about the sebaceous cyst or lipoma which the layman fears to be a cancer. On the other hand, some small glandular enlargement which the layman would ignore conjures up to us visions of leukæmia or lymphadenoma, and when we over-smoke we suspect carcinoma of the larynx, and so on. On the whole I think the doctor has the worse time when he is ill, and we must remember this when we doctor doctors.

Doctors' relatives again form a problem apart, and seem to make a rule of contracting odd diseases which seldom run a straightforward course. Once more you will agree with me that no doctor should undertake the professional care of any near relative, except for trivial ailments. And when we entrust the care of our wife or child to a colleague we should leave the patient entirely in his charge. We have all of us suffered many things from anxious medical relatives who constantly butt in with suggestions which do not make our task any easier. There are even doctors who insist on being present when their wives are being operated on, to the extreme discomfiture, no doubt, of the surgeon.

* Purvis Oration delivered to the West Kent Medico-Chirurgical Society on Dec. 10th, 1937.

THE DOCTOR IN A HURRY

To revert to my old doctor mentor, there were sundry other hints which he gave me, and some of them stuck in my memory. They are of such an elementary nature that I hesitate to retail them to this audience; but I venture to do so because not every doctor seems to have been similarly advised. For instance, he told me that there are few things which a patient dislikes more than a feeling that the doctor is in a hurry. "Never go into a patient's bedroom with your overcoat on," he said, "this will make the patient think you mean to be off in a minute or two. However over-worked and pressed for time you may be, don't let your patient sense it. Let him feel that, however short the consultation may have to be, during that period of time the nature of your patient's illness and how to get him well are the only things which matter to you." His last, and perhaps his best, bit of advice was "Never sit on the patient's bed." Those of us who have been seriously ill and have endured 12 stone of doctor on the edge of our bed will admit that he was right, often though most of us break this rule. But if you *must* sit on the patient's bed, for Heaven's sake don't idly turn over the pages of his *Daily Mirror* while he pours forth his tale of woe—an offence which I assure you I have seen committed more than once.

PROFESSIONAL CONTACTS

But if the general practitioner has the advantage over the consultant in respect of contact with his patients, the consultant is more fortunate in his opportunities of contact with other doctors, and this, you will all agree, is at the same time one of the most pleasant and one of the most important features of our professional lives. When we doctors meet we all of us learn something, and we rub one another's angles off. It is desirable that our meetings should not be too specialised and that general practitioners and consultants should meet on common ground. Also it is particularly desirable that specialists should commune not only with specialists, and finally that the young and the elderly should take counsel together. Certainly the elderly can learn much from the young, and even the most up-to-date young doctor can sometimes pick up a few wrinkles from an older colleague. As I have already said, all this is easy for the consultant, in fact it is almost forced upon him. He meets his colleagues of his own, and perhaps of other hospitals, almost daily. He learns from his house physician and his registrar. Various medical societies, medical dining clubs, or travelling clubs are available to him, and he would be foolish indeed not to embrace every opportunity so offered. But for the suburban, or still more for the country practitioner, such varied professional contacts are not available. Of necessity he must often lead a life of comparative professional isolation, though the modern tendency for the formation of firms of general practitioners with differing professional bents has done much to mitigate this. Fortunately the local medical society comes to his rescue, and on it he must rely for the catholic professional contact which should be such an invaluable factor in his career. Happy is the doctor, be he consultant or general practitioner, who has the chance of joining such a virile and enlightened association as the West Kent Medico-Chirurgical Society. When Dr. Prior Purvis founded this society in 1856 he raised up for himself a lasting monument, for so long as there are doctors in Kent this society will continue to flourish, and every year his name will be commemorated, however unworthily, in the Purvis Memorial Lecture.

MANAGEMENT OF THE NEUROPATH

Many years ago the St. Thomas's Hospital Medical and Physical Society, a club of medical students, invited me to open a debate on a topic of my own choice. The topic which I suggested was "That in the interests of public health the medical profession should be abolished." Briefly, my thesis was that if there were no doctors many patients would die as a result of peritonitis, or obstruction, or abscesses, who might have been saved by timely operation; fractures would not be set, nor ruptures cured; malaria and syphilis would not be adequately treated, and so on. But, I maintained, these items on the debit side would be heavily outweighted by those on the credit side. What a much happier and healthier world this would be, I urged, if the sphygmomanometer had never been invented, and if nobody knew what his blood pressure was! How humanity would gain if "dropped stomachs" and "colitis" had never been heard of!

I did not, of course, mean to be taken seriously in this piece of fooling. My object was to bring home to those young men what an infinity of harm the doctor may do when he attempts to explain neurotic symptoms on a non-existent organic basis. Every experienced general practitioner will admit that most of his time is spent in dealing with psychoneuroses. Thirty per cent. of the patients whom I see in consulting practice have, to the best of my belief, no organic lesion. Yet how many of us have the courage to tell our neuropathic patients what is really the matter with them? Very few, I fear. Recently I saw a woman of 38 who had been "delicate since childhood." The doctors had always been fighting her anæmia, she said. She had undergone deep X ray treatment for facial neuralgia, and when this failed, after a period of partial relief, ganglionectomy had been proposed, but fortunately declined. Naturally, her antra had been punctured and her septum straightened. Her spine had been manipulated by an orthopædic surgeon. Then suprarenal deficiency, long unrecognised, had at last been detected, and for a time suprarenal gland by the mouth had seemed to promise a cure. But alas the improvement was short-lived, and soon rapid heart action dominated the scene. Another doctor discovered that it was not defective suprarenal but rather over-active thyroid which was at fault, and iodine was administered. Finally an enterprising gynæcologist ascribed all her troubles to a retroverted uterus, performed ventral fixation and, en passant, removed her appendix. But she still suffers.

Now, this incredible story is almost a commonplace. Most of you can probably recall many analogous cases. This unfortunate young woman must have been seen by scores of doctors in various parts of this and other countries. Yet, she assures me, never a one had attempted to explain her many symptoms on the very obvious psychogenic basis.

On Sept. 23rd, 1518, Henry VIII wrote as follows in his Charter to the Royal College of Physicians: "Whereas, we consider it the duty of our Royal office by all means to consult the happiness of the people under our rule, we have thought it to be chiefly and before all things necessary to withstand in good time the attempts of the wicked, and to curb the audacity of those wicked men who shall profess medicine more for the sake of their avarice than from the assurance of any good conscience, whereby

many inconveniences may ensue to the rude and credulous populace." In the year 1937 we must confess that there are still to be found wicked men "who profess medicine more for the sake of their avarice than from the assurance of any good conscience," but I am glad to believe that, in this country at any rate, such men are few and far between. Many of the doctors whom this unhappy lady consulted were no doubt quite honestly convinced that they had discovered an organic cause for her symptoms, and that their treatment of it was rational. But I fancy that the majority of them realised at once that her troubles were psychogenic, but did not dare to say so.

What is the explanation of this discreditable state of affairs? I believe it is that many of us are still obsessed by the idea that our patients will decline to accept a psychogenic explanation for their symptoms. We know that they are often ashamed of such an explanation and are apt to resent it. Whereas the abdominal neurote will positively brag about his "dropped stomach," and proudly exhibit some horribly uncomfortable and entirely superfluous abdominal support; and the cardiac neuropath will excite sympathy from his relatives and friends by discoursing on his "tired heart" and the relief obtained from that magical drug digitalis. But we all know only too well that the improvement following such specious treatment is but short-lived. Sooner or later our patient will relapse, though possibly with quite different symptoms. Time was when the doctor who plucked up his courage and told a neuropath that his undoubted ill health was not due to any organic disease invariably elicited the indignant response "Do you mean to tell me that it is only nerves!"—and lost his patient. But I believe it to be true that nowadays patients are far more ready to accept a psychogenic explanation of their symptoms than they were some years ago. Most educated people have learnt that emotional upsets and conflicts can play havoc with our digestive processes, or with our circulation, for example. They are beginning to realise, too, that there are very few of us who do not from time to time suffer from disagreeable symptoms attributable only to nervous stresses of one kind or another, and that they are not "frauds" (to use their favourite word) when the dread of cancer engenders an anxiety state. It is, however, still very difficult to disabuse them of the idea that a neurosis is something to be ashamed of—an error which unfortunately is shared by some doctors.

I suggest to you, then, that we should be a little more courageous, and—may I venture to say it?—a little more honest, when dealing with neuropathic patients. The explanation of the true state of affairs must of course be done tactfully. It is futile to tell them that there is nothing the matter with them, and that they must pull themselves together and ignore their symptoms; and, in any case, this is not true. For instance, never tell a cardiac neuropath that he has a "tired," or a "toxic" heart, or that he has "a murmur which does not matter much." He will go away and brood over such statements, and wonder how long it will be before dropsy, or sudden death, occurs. Tell him with all the conviction you can that there is nothing wrong with his heart, as a heart. My experience is that these patients are enormously relieved at this news. I sometimes tell them that they are suffering from exactly the same most uncomfortable sensations as I experience when I go to look for a burglar, or when I begin to address a meeting such as this, and that these sensations are just as much out of my control as are their sensations. I explain to them that in both cases the palpitations and precordial discomfort are the result of emotional disturbances, which differ only in kind and in degree.

THE BLOOD PRESSURE OBSESSION

And now for a word or two about that widespread disorder of modern times which one may call the blood pressure obsession. I suggest to you that it is a mistake to take blood pressures too frequently, and that the patient should never be given any figures. I understand that there is one distinguished physician who feels so strongly on this point that he flatly declines to take the blood pressure of any of his patients. But perhaps this is carrying things too far! May I remind you, too, that blood pressures are often very variable, from day to day or even from minute to minute, in the same patient, and that in different individuals the normal pressure varies within wide limits. It is a fallacy that the blood pressure at age 40 should be 140, at age 50 150, and so on. In point of fact the average blood pressure at age 20 is 122, at age 35–40 125, and at age 55–60 133. Moreover, let me stress the fact that these pressures are *averages*, and that the normal pressure varies between 100 and 160, systolic. Owing to a misconception of the true facts of the case I venture to assert that a large proportion of the population are at present firmly convinced that their blood pressures are too low, and suffer accordingly. I assure you that for some time past whenever I begin to apply the armlet to my patients they almost invariably remark, "I'm afraid you will find it too low, doctor!" The subsequent finding being 110 or even 120, and therefore perfectly normal.

THE PATIENT'S ATTITUDE TO THE DOCTOR

And now to pass from doctors to patients. I have often wished that someone would publish a small manual entitled "Advice to Patients about to Consult their Doctor"—though I doubt whether such a pamphlet would be widely read, or, if read, acted upon. But what a lot of valuable time would be saved if our patients could be taught that all we want to hear from them is an account of their symptoms, as concise as possible and chronological! Also we want to know something of their daily routine, what time they get up and go to bed, the mental and physical stresses to which they are subjected, and their habits as to food, drink, and tobacco. What we do *not* want to know are the very things which they are just bursting to tell us—e.g., what Dr. X of Freiberg or Prof. Z of Lausanne thought about them.

The patient who particularly infuriates me is the one who, when asked what his trouble is, produces a large bundle of, usually, very mediocre X ray films, and says, "There are Dr. Blank's X rays." Almost worse is the one who reverently deposits on my desk a gigantic dossier of pathological reports, and looks quite injured when told that these will be looked at presently, after he has described his symptoms. Then there still exist patients who expect us to diagnose their illness without a complete physical examination of their person. My old doctor mentor used to say, "Insist that every one of your male patients takes off his shirt, collar, and vest, and that every female patient takes off her corsets." (Ladies *did* wear corsets in those days!) I have often thought that it is less difficult for a camel to enter the eye of a needle than it is to persuade a lady to remove her abdominal belt; but persuade her we must, or sooner or later we will endure the mortification of

finding that we have failed to discover the existence of an unsuspected fibroid or something of the sort.

Then I would like to have our patients taught how to choose their doctor, and having chosen him to be loyal to him, in their own interests. I admit at once how difficult this is for them. Whenever they fall ill and do not speedily recover, they are importuned incessantly by well-meaning but misguided friends "Do see *my* doctor; he is too *marvellous*," or, more frequently, "Go straight to Prof. Blank at Berlin. The German doctors are years in advance of ours." (This cult of foreign doctors is, of course, a survival of witchcraft; one sees exactly the same thing when, in the hospital, the Lambeth patient tells you, with bated breath, what treatment a "black doctor" advised.) But when loyalty to the doctor is stretched to the breaking-point and confidence has gone, it is far better for the patient, and kinder to the doctor, to come out into the open and say so, and choose another doctor. It is quite true that in sickness "where no counsel is the people fall," but one must alter the rest of the quotation—"In a multitude of counsellors there is *not* wisdom." My heart often bleeds for the poor rich man when I see him surrounded on his sick-bed by some half a dozen doctors, and reflect how very much more fortunate is he who is unable to afford such a very dubious luxury.

"AN INDEPENDENT OPINION"

This brings me to the question of "a second opinion." And here we will all be on common ground when I say that it is very natural that the patient or his relatives should ask for further advice in serious or obscure illness. It is bound to be a little galling to any doctor when a consultation is requested though all is going well and he feels quite competent to deal with the situation. But it is, or should be, a first principle with every doctor to forestall his patient in asking for a second opinion when things are not going well. The general practitioner who gets the reputation of constantly demanding a consultation naturally soon forfeits the confidence of his patients. But I think we should always ask for further advice when in doubt as to diagnosis and treatment. And in cases of dangerous illness, even if we feel that the diagnosis is only too clear and that all treatment is futile, it is usually wise to explain the situation to the relatives, to state our conviction that nothing more can be done, but at the same time to ask them if they would like further advice, so that afterwards they can feel that everything possible had been done.

Then comes the question of choice of consultant. And here we are usually up against a brick wall. It seems to be impossible to disabuse the patient and his relatives of the idea that the consultant will be "biased" by the general practitioner. I do wish that it were possible to bring home to our patients that no reputable consultant will be biased by any other doctor, and that the best judge as to who is the right consultant for any particular patient is that patient's family doctor! The patients who sneak off to consultants behind their doctor's back are not doing the best for themselves. They crave for "an independent opinion"; but only too often they are unfortunate in their choice of consultant, to put it mildly. And the consultant is hampered in his work owing to the fact that important information, which only the general practitioner could give him, is being withheld. Finally, when this perhaps unsatisfactory consultation is over, any treatment which the consultant can suggest can be carried out only under the supervision of the general practitioner, who is apt to be incensed when he hears what has happened. But, as I hinted before, this is one of the frailties of human nature which must be accepted and made the best of. We must all of us be prepared for this craving of the average patient for the so-called "independent" opinion. We must just try to visualise his point of view, and, if we can, to sympathise with it; but this is sometimes very difficult.

CARRYING HONESTY TO EXTREMES

Another understandable, but nevertheless regretable, foible of our patients is their liking for elaborate investigation and spectacular treatment; but we must be very careful not to pander to this. I feel that there is a tendency for some transparently honest doctors to be carried away by their enthusiasm for what they conceive to be up-to-date scientific doctoring; they run through the whole gamut of laboratory investigation, hæmatological, bacteriological, biochemical, and radiological, where to the, shall we say, more prosaic, doctor there seems to be little indication for anything of the sort. The subsequent treatment is equally elaborate. It nearly always includes frequent colonic lavage, and various dietetic restrictions which seem to have no scientific basis. I need hardly add that vaccines or other injections—calcium is for some inscrutable reason the fashionable one at the moment—are almost a sine qua non. It cannot be denied that most patients, and even the very intelligent ones, are impressed by this sort of thing, and are apt to think that the doctor who does not indulge in these antics is not progressive. Wisdom usually comes to them ultimately through bitter experience; but at what a cost!

We must not go to the other extreme and become altogether nihilistic in the treatment of conditions for which we know of no specific therapy. It is a difficult thing, and perhaps an unkind deed, to tell the mother of a child dying of acute leukæmia that her child will most certainly be dead within a fortnight, and that no treatment will be of any avail. In such a tragic contingency I think it is justifiable to attempt some sort of treatment, even though you know it to be hopeless. I have never forgotten a young man with retrobulbar optic neuritis whom I sent to a well-known and very honest, perhaps you will say too honest, neurologist. He told the patient's mother that he did not know the cause of the optic neuritis; that the patient might go blind, or, alternatively, might recover; and that no treatment would be likely to help. This was, I think, carrying honesty to extremes. Needless to say the patient was taken to Zurich, where blisters were applied to the temples, and normal saline was injected in minim doses under the conjunctiva. Also, needless to say, to this day the mother is firmly convinced that only her prescience in taking her son to Zurich brought about the recovery of vision which so happily ensued! Still, though there is such a thing as being too honest, as in the instance just related, it is never to be forgotten that honesty is the best policy in doctoring as in everything else. It may sometimes be wise in the patient's interests not to disclose the full facts, but he should never be deceived.

"THE DOG SHOULD NEVER HAVE DIED"

Before I close I have a confession to make. It is this. When my dog is ill I behave like the most abject of patients. If one vet. does not quickly cure him I take him to another, and if necessary to a third, or even a fourth. I pour down his unwilling

throat all the patent medicines and nostra which my doggy friends recommend. And finally, after he is dead I find myself telling everybody, "I'm sorry to say it, but the case was badly mismanaged by the vet. The dog should never have died."

SEROTHERAPY IN LEPROSY

THE serum that Prof. Reenstierna has prepared for the treatment of leprosy is obtained from sheep which have been previously inoculated with partially acid-fast organisms. The organisms used are the well-known Kedrowsky strain and a new strain, morphologically and culturally similar, recovered from a leprous patient's blood. Carbolic acid is added to a strength of 0·5 per cent., and after storing in a refrigerator for a fortnight the serum is tested for contamination.

The remedy has been tried on 65 cases of leprosy in Addis Ababa, on 53 in Columbia, and on 30 in Venezuela (*Acta med. scand.* 1937, Suppl. lxxxv). After its use nodules became flatter and softer and the skin covering them became wrinkled; the brownish colouring of the skin changed to normal; cutaneous ulcers of the face and extremities healed rapidly; nasal obstruction was relieved and ulcers healed; anæsthesia, neuralgia, paralysis of the hands, œdema, and fever all diminished. The patients each received three intragluteal injections of 10 c.cm. at two or three days' intervals, followed by another similar course after a month. Reenstierna gives details of the South American cases, with illustrations of improvement in 3, but no record is made of any diminution in the number of bacilli in the body. It is unfortunate also that a control was not arranged —e.g., by treating half the patients with serum of uninoculated sheep. This control would have been easy to carry out and an obvious cause of criticism might have been removed.

Reenstierna does not offer any suggestion as to how the serum acts. In closing he admits that it is still too early to give an opinion of the effects and that a cure through serotherapy alone is highly improbable. "But," he adds, "the rapid and favourable action of the antileprosy serum, observed by more than 20 physicians in different countries, on leprous manifestations on which injections of chaulmoogra oil and its derivatives have had little or no effect, entitles me to the assumption that treatment with antileprosy serum, carried out in the most advantageous manner, ought to be able to become a good auxiliary to the standard therapy with chaulmoogra."

A STUDY OF RACIAL DIFFERENCES

WHILE some believe that there are real and innate differences between the various races of mankind, others hold that these differences are superficial, depending not upon nature but upon nurture. When issues become political, as this one has, partisanship is apt to hinder the search for truth; and although both these creeds are stoutly, and often acrimoniously, defended, neither has been scientifically corroborated. Prof. S. D. Porteus sets out not to prove a preconceived theory but merely to collect data and draw what conclusions he may.[1] As he points out, the term "race" is only properly applicable to certain geographically and culturally isolated groups of people in which a common physical type has been established as a result of inbreeding and various factors of natural selection. If such groups are compared by means of mental tests and by a study of their environment, social adaptability, and educability, reliable evidence of relative superiority or inferiority may be obtained. For his present purpose Prof. Porteus chose the Bushmen of South Africa and two groups of Australian aborigines and found that, despite a less favourable environment, the Central Australian aborigines were in almost every respect superior to their more happily situated neighbours of the North West. No less interesting is his conclusion that the Bushmen of the Kalahari, though undoubtedly enjoying a better environment than the Central Australian aborigines, were less educable and inferior in planning ability, but superior in physical courage and artistic skill. This book is not merely a scientific treatise for the initiated; it is also a delightful book of travel.

[1] Primitive Intelligence and Environment. By S. D. Porteus, D.Sc. London: Macmillan and Co., 1937. Pp. 325. 15*s*.

PROTECTION AGAINST MEASLES

Dr. C. F. McKhann of Boston has been interested for some time in the use of placental extract in the prevention and modification of measles, and he has recently compared its effects with those of adult and convalescent serum.[1] The groups observed were fairly large, probably enough to be statistically significant. Complete protection was given with adult serum to 56·4 per cent., with convalescent serum to 75·4 per cent., and with placental extract 56·3 per cent., while the severity of the attack was modified in 23·8, 16·8, and 30·4 per cent. respectively. The placental extract appeared to be the most successful, with only 5·3 per cent. failures, followed by convalescent serum with 7·8 per cent. and adult serum with 19·8 per cent.

As McKhann points out, although there has been a steady decline in the death-rate from measles, this is believed to be due largely to general measures such as recognition that it is a dangerous disease, prevention of needless exposure, isolation of patients to combat secondary infections, and better child health and nutrition. Further improvement is desirable, but in employing such immunisation methods as these it must be understood that complete protection through the use of human immune bodies lasts only a few weeks. This is of particular advantage only when used in institutions or for acutely or chronically ill, debilitated, or very young children. Otherwise it is better to aim for the permanent protection given by an attenuated attack. This may be achieved by injecting the immunising agent either in full doses in the second half of the incubation period or in proportionately smaller amounts as soon as possible after exposure.

[1] *J. Amer. med. Ass.* 1937, **109**, 2034.

NEW PREPARATIONS

"NESTROVITE" BRAND VITAMIN CONCENTRATE.—Roche Products Ltd. (51, Bowes-road, London, N.13) have put on the market Nestrovite Emulsion and Nestrovite Tablets, accessory food preparations evolved in the laboratories of Roche and Nestlé and made in Great Britain. These contain vitamins A, B_1, C, and D in a palatable base, and for each vitamin the quantity present is stated precisely in international units. Two tablets (the usual dose for an adult) is regarded as "equivalent to approximately one tablespoonful of medicinal cod-liver oil, two tablespoonfuls of orange juice . . . and an ample allowance of vitamin B_1." The emulsion is recommended for infants, and the use of one or other of the two preparations is advised throughout childhood and in a variety of disorders in adults for which vitamin deficiencies have been held responsible.

"WELLCOME" BRAND CALCIUM MANDELATE COMPOUND.—The use of calcium (rather than sodium or ammonium) mandelate in the treatment of urinary infections was proposed by E. Schnohr in THE LANCET last year (1937, **1**, 1104). The preparation now put out by Burroughs Wellcome and Co. (Snow Hill-buildings, E.C.1) is a powder of which 4·4 grammes ("the normal adult dose") is equivalent to 3·0 g. of mandelic acid. The chief advantage claimed for the calcium salt is that it causes no nausea and only negligible digestive disturbance.

SULPHANILAMIDE TABLETS G.L.—Glaxo Laboratories (Greenford, Middlesex) are now manufacturing

a preparation of *p*-aminobenzenesulphonamide (sulphanilamide), in tablets containing a quarter of a gramme and half a gramme.

We are asked to state that the London address of ORGANON LABORATORIES has been changed from 1, Gordon-square, W.C.1 to 77, Newman-street, W.1. The telephone number at the new Newman-street house will be Museum 2857 as previously.

THE BRITISH DRUG HOUSES LTD. have now completed their laboratories for the preparation of B.D.H. sera and vaccines and have produced an illustrated booklet containing notes on indications, administration, dosage, packings, and prices, together with a short bibliography of vaccine and serum therapy. This will be sent to readers applying to Graham-street, City-road, N.1.

Vacancies

For further information refer to the advertisement columns

Aberdeen University.—Lecturer in Bacteriology, £500–£600.
Army Dental Corps.—Dental Surgeons.
Auckland Hosp. Board.—Radiotherapist and Radiologist, each £1000.
Belgrave Hosp. for Children, Clapham-road, S.W.—Asst. Physician, 50 guineas.
Bethnal Green Metropolitan Borough.—Dental Anæsthetist, 3 guineas per session.
Birmingham, Children's Hosp.—Receiving Room Officer, £250. Res. Sen. Cas. H.S., £125. Also First Asst. to Orthopædic Dept., £100.
Birmingham City Education Committee.—Asst. School M.O., £500.
Blackburn, Brockhall Certified Institution for Mental Defectives, Langho.—Deputy Med. Supt., £750.
Blackburn County Borough.—Asst. M.O.H. and Asst. School M.O., £600.
Bootle General Hosp.—H.P., two H.S.'s, and Cas. O., each at rate of £150.
Cape Town University.—Sen. Asst. in Dept. of Anatomy, £800.
Cheshire, St. Anne's Home, Altrincham.—Res. Surg. O., £200.
Chesterfield, Walton Sanatorium.—Jun. Res. Asst. M.O., £350.
Dartford Rural District Council.—Part-time M.O., at rate of £250.
Ealing Borough.—Asst. M.O., £600. Res. Asst. M.O., £350. Also Ophth. Surgeon, £275.
Eastbourne, Princess Alice Hosp.—Res. H.S., £150.
East Suffolk County.—M.O.H. for Woodbridge Urban and Deben Rural Districts and Asst. County M.O.H., £776.
Evelina Hosp. for Sick Children, Southwark, S.E.—H.P., at rate of £120.
Gloucestershire Royal Infirmary.—Res. Surg. O., £200.
Grimsby and District Hosp.—Sen. and Jun. H.S.'s, at rate of £200 and £150 respectively.
Halifax Royal Infirmary.—Radiologist, £700.
Hampstead General and N.W. London Hosp., Haverstock Hill, N.W.—H.P. and H.S., each at rate of £100.
Horton General Hosp., Banbury.—Res. M.O., at rate of £150.
Hosp. for Sick Children, Great Ormond-street, W.C.—Asst. Pathologist, £400. Cas. M.O., at rate of £175. Also Two Anæsthetists, each 15 guineas.
Huddersfield, St. Luke's Hosp.—Res. M.O., £230.
Ipswich, East Suffolk and Ipswich Hosp.—Cas. O., £144.
Islington Metropolitan Borough.—Asst. M.O., £600.
Jewish Maternity Hosp., Underwood-street, E.—Res. Med. O., at rate of £50.
Keighley and District Victoria Hosp.—Second Res. M.O., at rate of £120.
King's College Hosp., Denmark Hill, S.E.—Asst. Physician.
Leeds City.—Asst. M.O., £500.
Leicester Royal Infirmary.—Res. Anæsthetist, at rate of £150.
Liverpool, Alder Hey Hosp.—Res. Asst. M.O. for Orthopædic Wards, at rate of £200.
Liverpool City.—Res. Asst. M.O.'s for Special Hospitals, each £200.
Liverpool County Mental Hosp., Rainhill.—Asst. M.O., 7 guineas per week.
Liverpool Dental Hosp., Pembroke-place.—Hon. Anæsthetist.
London Chest Hosp., Victoria Park, E.—H.P., at rate of £100.
London County Council.—Asst. M.O.'s, Grade 1, each £350. Also Temp. Part-time Asst. M.O.'s for School Medical Work, each 30*s.*
London Hosp., E.—Surgical First Asst. and Reg., £300.
Maidstone, Kent County Ophthalmic and Aural Hosp.—Ophth. H.S., at rate of £200.
Maidstone, Kent County Mental Hosp.—Asst. M.O., £509.
Maidstone, West Kent General Hosp.—H.P., £175.
Manchester, Ancoats Hosp.—Pathologist and Radiological Officer, each £400. Also Cas. O., £250.
Manchester, Christie Hosp. and Holt Radium Institute, Withington.—Res. Surg. O., at rate of £150.
Manchester Northern Hosp., Cheetham Hill-road.—Res. H.P. and Res. H.S., each at rate of £100.
Metropolitan Hosp., London, E.—Hon. Anæsthetist, £25.
Middlesex Hosp., W.—Hon. Physician for Dept. of Psychological Medicine.
Ministry of Pensions, London, S.W.—Medical Officers, £750.
National Hosp., Queen-square, W.C.—H.S., £100.
National Temperance Hosp., Hampstead-road, N.W.—Hon. Anæsthetist. Also Med. Reg., 40 gns.
Newcastle-upon-Tyne, Royal Victoria Infirmary.—Hon. Surgeon.
Persian Gulf, Bahrain Government.—M.O., 1400 Rs. per month.
Poplar Hosp. for Accidents, East India Dock-road, E.—Second Res. O., at rate of £175.
Portsmouth Royal Hosp.—H.P., H.S. to Orthopædic Dept., and Cas. O., each at rate of £130.
Preston and County of Lancaster Royal Infirmary.—Res. Anæsthetist, at rate of £200.
Preston and County of Lancaster Royal Infirmary.—M.O. to V.D. Dept., £350.
Preston, County Mental Hosp., Whittingham.—Res. Jun. Asst. M.O., £500.
Queen's Hosp. for Children, Hackney-road, E.—H.S. and Cas. O., each at rate of £100.
Rotherham, Alma-road Hosp.—Obstet. M.O. and Deputy Med. Supt., £750.
Royal Masonic Hosp., Ravenscourt Park, W.—Res. Surg. O., at rate of £250.
Royal National Orthopædic Hosp., 234, Great Portland-street, W.—Two H.S.'s for Country Branch, each at rate of £150.
Royal Naval Medical Service.—M.O.'s.
Ruthin Castle, North Wales.—Res. Physician, at rate of £200.
St. Leonards-on-Sea, Buchanan Hosp.—Hon. Surgeon.
Sheffield, Jessop Hosp.—Res. Anæsthetist, at rate of £100.
Sheffield Royal Infirmary and Royal Hosp.—Two First Assts. to Fracture Clinics, each £300.
South Eastern Hosp. for Children, Sydenham, S.E.—Hon. Pathologist, £50.
South Shields, Ingham Infirmary.—Jun. H.S., £150.
Surrey County Council.—A.M.O., £600.
Taunton and Somerset Hosp.—H.S., £125.
Torquay, Torbay Hosp.—H.P., £175.
Ventnor, Royal National Hosp. for Consumption, &c.—Second Asst. Res. M.O., at rate of £200.
Wakefield, Scotton Banks Sanatorium.—Sen. Asst. M.O. and Deputy Med. Supt., £450.
Walsall Manor Hosp.—Ear, Nose, and Throat Surgeon, £200.
Wolverhampton Royal Hosp.—H.S., at rate of £100.
The Chief Inspector of Factories announces vacancies for Certifying Factory Surgeons at Morley (Yorks, W.R.), Thirsk (Yorks, W.R.), Acton and Ealing (Middlesex), Barking (Essex), Carnforth (Lancashire), Kibworth (Leicestershire) and Ystalyfera (Glamorgan).

Appointments

BANHAM, A. R., M.B. Camb., F.R.C.S. Eng., Hon. Surgeon to the General Hospital, Northampton.
BINNING, REX, M.R.C.S. Eng., Clinical Assistant Anæsthetist to the Royal Sussex County Hospital, Brighton.
BIRDSALL, S. E., B.Chir. Camb., F.R.C.S. Eng., Hon. Laryngologist to the Woolwich and District War Memorial Hospital.
BRUCE-JONES, D. B. S., M.D. Durh., Hon. Physician to the Skin Department, Paddington Green Children's Hospital.
COLLEDGE, LIONEL, M.B. Camb., F.R.C.S. Eng., Hon. Assistant Surgeon to the Ear, Nose, and Throat Department of the Prince of Wales's General Hospital, London.
CREER, W. SAYLE, M.Ch.Orthop. Liverp., F.R.C.S. Eng., Orthopædic Surgeon-in-Charge of Fractures to the District Infirmary, Ashton-under-Lyne.
GARLAND, T. O., M.B. Camb., D.P.H., Medical Officer of Health for Kettering.
GOLDSMITH, A. J. B., M.B. Lond., F.R.C.S. Eng, Hon. Ophthalmic Surgeon to the Paddington Green Children's Hospital.
HUTCHESON, A. S., M.B. Glasg., Assistant Medical Officer of Health for Argyll.
JAMIESON, JANET M., M.B. Glasg., D.P.H., Maternity and Child Welfare Officer and Chief Supervisor of Midwives for Northumberland.
JENKINS, MARGARET W., M.B. Edin., Medical Officer at Winford Orthopædic Hospital, Bristol.
LAURENT, L. J., M.D., M.R.C.P. Lond., Deputy Medical Superintendent (Grade II) at the Eastern Hospital L.C.C.
MASON, G. A., M.B. Durh., F.R.C.S. Eng., Hon. Assistant Surgeon to the Royal Victoria Infirmary, Newcastle-upon-Tyne.
MILLS, EDWARD, M.B. St. And., D.P.H., Assistant Medical Officer of Health for the County of Inverness.
MONRO, A. B., M.B. Edin., D.P.M., Second Assistant Medical Officer at the County Mental Hospital, Rainhill.
MUMFORD, P. B., M.D. Manch., F.R.C.P. Lond., Hon. Consulting Dermatologist to the Christie Hospital and Holt Radium Institute, Manchester.
PORTER, ARTHUR, M.D. Camb., M.R.C.P. Lond., Out-patient Medical Registrar at St. John's Hospital for Diseases of the Skin, London.
SAVATARD, LOUIS, L.S.A., Hon. Consulting Dermatologist to the Christie Hospital and Holt Radium Institute, Manchester.
SOLLEY, RUPERT, M.B. Camb., Assistant Medical Superintendent at Exeter City Mental Hospital.
STEVENSON, D. C. L., F.R.C.S. Edin., Resident Surgical Officer at the Woolwich and District War Memorial Hospital.
TREBLE, H. A., B.M. Oxon., M.R.C.P. Lond., Assistant Physician to the Kent and Canterbury Hospital.
Middlesex Hospital.—The following appointments are announced:—
WILSON, C. P., F.R.C.S. Eng., Aural Surgeon;
MONKHOUSE, J. P., M.B. Lond., F.R.C.S. Eng., Assistant Aural Surgeon;
RIVETT, L. C., M.A. Camb., F.R.C.S. Eng., F.C.O.G., Obstetric Surgeon; and
WINTERTON, W. R., M.B. Camb., F.R.C.S. Eng., M.C.O.G., Assistant Obstetric Surgeon.